Second Edition

Urgent Care Emergencies

Avoiding the Pitfalls and Improving the Outcomes

Edited By Deepi G. Goyal and Amal Mattu

WILEY

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SECOND EDITION

EDITED BY

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Preface to the Second Edition

Injuries and illnesses are unpredictable. Affected patients want and deserve timely and effective care.

In the decade since the publication of the first edition of *Urgent Care Emergencies: Avoiding the Pitfalls and Improving the Outcomes*, the healthcare landscape has continued to evolve dramatically, with the surging demand for alternative venues to address unscheduled injuries and medical conditions. Healthcare systems remain stressed with rising patient volumes, staffing instability, and increasing cost constraints. As traditional emergency departments and primary care offices are overwhelmed, walk-in clinics, urgent care, and express care centers have emerged as vital components of the healthcare system to allow patients timely access for lower-acuity conditions.

Since the original publication in 2012 there has been substantial growth of urgent care centers and similar facilities. According to the Urgent Care Association, the number of urgent care centers in the United States has increased by over 50% in the past decade, with more than 14,000 centers now in operation. This expansion reflects the increasing reliance on these facilities to provide accessible, efficient care for noncritical conditions. The role of urgent care centers in alleviating the burden on emergency departments and improving patient outcomes is more critical now than ever.

Because most patients coming to these settings have low-acuity presentations and diagnoses, providers must be even more vigilant to avoid errors that may lead to poor outcomes and simultaneously identify high-risk diagnoses that should be expeditiously referred to other settings.

This second edition of *Urgent Care Emergencies* seeks to address the challenges faced by healthcare providers in these dynamic environments. It underscores the importance of timely and accurate diagnosis and treatment of seemingly minor injuries and illnesses to avoid serious morbidity. The text aims to highlight both common pitfalls in the management of low-acuity conditions and high-risk diagnoses that must be excluded. This edition includes a chapter addressing medical malpractice, especially as it pertains to medical practice in the United States.

All chapters in this edition have been completely updated to reflect the latest clinical guidelines and best practices. The chapter authors were selected for their expertise in their respective fields, ensuring that the content is both authoritative and relevant. At the end of each chapter readers will find valuable pearls of wisdom aimed at enhancing patient care and outcomes.

While the title suggests that this book is aimed at providers in urgent care settings, it is meant to serve as a valuable resource for any healthcare provider who evaluates low-acuity complaints, whether in an emergency department, urgent care center, clinic, or other healthcare setting. It is not designed to be comprehensive but is meant to be practical, suitable for cover-to-cover reading or as a reference during daily practice. We hope that this updated edition will continue to support healthcare professionals in delivering the highest-quality care to their patients.

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CHAPTER 1
HEENT Pitfalls

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Introduction

Emergencies affecting the head, eyes, ears, nose, and throat (HEENT) constitute a large component of chief complaints seen in urgent care centers. The majority of these patients have benign conditions that can be managed on an outpatient basis. However, some seemingly innocuous complaints can portend more serious diseases that pose a significant risk of morbidity and possibly mortality. As with most diseases, the key to differentiating between minor and dangerous conditions is the history and physical examination. A huge spectrum of pathology can manifest in the head and neck. In this chapter we present key facts, highlight pitfalls in diagnosing these conditions, and offer pearls intended to facilitate their management.

Eye

Pitfall | Failure to identify red flag symptoms leads to inappropriate or unrecognized need to transfer patients requiring emergency evaluation

A wide variety of eye complaints are encountered by urgent care providers. Identifying the emergent conditions that require emergency department (ED) or emergent ophthalmologic care is crucial. Red flags include a severely painful red eye, acute vision loss, anisocoria, and photophobia [1]. An acute red eye may represent acute angle glaucoma, endophthalmitis, uveitis, or keratitis. Visual acuity should be documented on these patients. Acute monocular visual loss may signify another dangerous condition, such as central retinal artery occlusion, central retinal vein occlusion, retinal detachment, or a central nervous system process (stroke, transient ischemic attack [TIA], or multiple sclerosis). Patients with concern for these diagnoses should be referred to an ED or for emergent ophthalmologic follow-up. After identifying which patients need a higher level of care, there are still many eye conditions that can effectively be cared for in the urgent care setting.

Corneal abrasion, ulcers, and foreign bodies

Differentiation between corneal abrasions, corneal ulcers, and corneal foreign bodies (FBs) can be difficult. The majority of patients with any of these conditions present with eye pain and a gritty or FB sensation. Patients with suspected corneal epithelial defects should have a full eye examination. Use of a slit lamp is preferred to the Wood's lamp. The instillation of analgesic and/or cycloplegic drops will significantly relieve the patient's symptoms and increase their ability to tolerate the examination, but these drops should not be used if globe rupture is suspected. Globe rupture can occur after trauma or a penetrating FB and should be suspected if there is buckling of the sclera or if there appears to be fluid draining from the orbit. This is a true ophthalmologic emergency, and no pressure or foreign material should be introduced into the eye of this patient. Fluorescein staining is mandatory for the evaluation of

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Table 1.1 Treatment of conjunctivitis, corneal abrasions, ulcers, and foreign bodies [3–6].

Indication	Contact lenses	Antibiotic	Dose	Duration of therapy
Abrasion or FB	No	Trimethoprim/polymyxin B	1–2 drops q 3 h	Continue until symptom free for >24 h
Conjunctivitis	No	Trimethoprim/polymyxin B	1 drop q 6 h	5–7 d
Abrasion or conjunctivitis Not from FB	No	Erythromycin	Ointment 0.5″ q 2–4 h	Continue until symptom free for >24 h/5–7 d
Abrasion or FB	Yes	Ciprofloxacin	Day 1–2: q 2 h when awake; days 3–7: q 4 h	Until evaluation by ophthalmologist
Conjunctivitis	Yes	Ciprofloxacin	1–2 drops q 6 h	5–7 d
Abrasion or FB	Yes	Moxifloxacin	1 drop q 6–8 h	Until evaluation by ophthalmologist
Conjunctivitis	Yes	Moxifloxacin	1 drop q 6–8 h	7 d
Ulcer	NA	Moxifloxacin	1–2 drops q 1 h	Until evaluation by ophthalmologist
VZV	NA	Antiviral: acyclovir or ganciclovir	IV/oral/topical	Ophthalmologist evaluation needed

FB, foreign body; IV, intravenous; NA, not applicable; q, every; VZV, varicella zoster virus.

a corneal defect and to assess for ulcers or dendritic lesions seen with herpes simplex virus (HSV) keratitis. Varicella zoster virus (VZV) should be suspected in patients with a vesicular rash on the forehead or nose and should be referred to ophthalmology [2]. In patients with herpes virus infection concerns (HSV or VZV), steroids should only be used if directed by ophthalmology. Defects in the epithelial surface appear as a stain that does not clear with blinking. The size and position of any defect(s) should be documented. Punctate defects, which appear in a circular pattern, are sometimes seen in contact lens wearers, particularly after prolonged wear. Larger defects with a crater formation are ulcers.

Parallel vertical abrasions should raise suspicion for an FB under the lid. When this type of injury is detected, the patient's eyelid should be everted to allow further assessment. Without treatment, and over time, the vertical abrasions will coalesce and form an ulcer. An FB may be superficially lodged directly on the corneal surface. In many cases FBs can be removed with a cotton swab or by irrigation, but if that procedure is unsuccessful, removal with a needle or burr may be necessary and should be done only by someone with specific training. Metal FBs may lead to the development of a rust ring, which should be removed by an ophthalmologist [3]. Emergent ophthalmologic consultation is indicated for globe rupture or intraocular foreign body. Following removal of the superficial FB, patients should be treated with antibiotic drops (not ointment) as per Table 1.1. An FB consisting of vegetation should be treated with a fluoroquinolone [4]. Perforating or intraocular FBs will require systemic antibiotics and transfer [3].

KEY FACT | **Patients with corneal abrasions, ulcers, and corneal foreign bodies should all be treated with antibiotics.**

The immediate treatment of corneal abrasions, ulcers, and FBs is similar (Table 1.1). Simple abrasions that are smaller than 3 mm do not require follow-up as long as an FB is not present, the patient's visual acuity is normal, and symptoms resolve within 24 hours [7]. However, if there is any doubt, referral to an ophthalmologist is reasonable. All other defects should be seen by an ophthalmologist within 24 hours. Antibiotics, which may be prescribed as either ointment or drops, should be administered to all patients with epithelial defects. Ointment is generally preferred (particularly for children) because it stays in place longer and lubricates the eye. However, ointments are not well tolerated by most adults because they obscure vision. Contact lens wearers with corneal abrasions require antipseudomonal antibiotic coverage and should be advised to refrain from wearing their contact lenses until they are cleared to do so by an ophthalmologist. All patients with corneal ulcers also require antipseudomonal antibiotic coverage.

Patients with painful corneal abrasions may require systemic analgesia. Ophthalmic nonsteroidal anti-inflammatory drugs (NSAIDs) may be prescribed but are expensive. Topical anesthetics such as tetracaine when used for the first 24 hours have been shown to improve analgesia, but should be used with caution as repeated use may be associated with the development of ulcers [8]. Eye patching has not been shown to be effective in accelerating healing. In fact, because it might worsen the infection and thus lengthen the time to recovery, patching is not recommended [9].

Conjunctivitis

Commonly known as "pink eye," conjunctivitis presents with an irritated, erythematous conjunctiva with discharge. Visual acuity should be minimally affected, if at all. The etiology may be infectious or noninfectious (allergic/chemical). Infectious causes are most commonly bacterial or viral, and it may be difficult to differentiate between the two. Generally, a thin watery discharge from the eye accompanied by symptoms of an upper respiratory infection is more likely to be viral in etiology. This pathology is self-limiting and patients do not require treatment with antibiotics. Symptom relief with cool compresses, artificial tears, and anticipatory guidance regarding duration of symptoms and handwashing are paramount to good patient care. Purulent drainage is more commonly caused by a bacterial source [10]. While bacterial conjunctivitis may improve on its own, topical antibiotics are recommended. Topical antibiotics typically shorten the duration of illness and decrease the amount of time out of work/school. Patients who wear contact lenses need to be treated with antipseudomonal antibiotics (see Table 1.1). Hyperpurulent discharge, frequently seen in neonates, immunocompromised patients, or patients with a history of sexually transmitted infection, is most concerning for gonococcal or chlamydia conjunctivitis, which requires systemic antibiotics. Testing/culture should be completed on these patients. Allergic conjunctivitis presents with mild erythema and itching as a primary concern. It can be treated with topical antihistamines or mast-cell stabilizers. Conjunctivitis rarely causes severe eye pain, constant visual loss, or photophobia. If these symptoms occur, consider an alternative diagnosis.

KEY FACT | **All patients with corneal ulcers require antipseudomonal antibiotic coverage.**

Hyphema versus subconjunctival hematoma

A hyphema is a collection of blood in the anterior chamber commonly occurring from trauma. Patients typically present with eye pain and pupillary constriction. Visual acuity is variably affected, based on the amount of blood present.

Patients who should receive emergent consultation and strong consideration of admission include those with sickle cell disease, bleeding dyscrasias (such as anticoagulant use or hemophilia), potential open globe injuries, elevated intraocular pressure (IOP), or hyphemas that fill ≥50% of the anterior chamber [6]. Healthy patients with none of the above risk factors can be discharged home with ophthalmologic follow-up within 12–24 hours. Patients should elevate their head and avoid bright lights. A rigid shield is sometimes used to avoid any further trauma. NSAIDs should be avoided because of their associated bleeding risk. Nausea and vomiting should be treated aggressively since they can raise IOP. These instructions should be clearly communicated to any patient being discharged. Patients suspected to be unreliable and those unable to return if their condition worsens should be observed or admitted.

Subconjunctival hematomas occur where there is blood that has extravasated from the conjunctival or episcleral vessels into the subconjunctival space. Subconjunctival hematomas are typically painless. They may occur after minor trauma or be atraumatic. They are more common in older individuals who have more fragile vessels (patients with diabetes, hypertension, hyperlipidemia, etc.) [11]. The appearance of subconjunctival hemorrhage can be dramatic and very concerning to the patient, but fortunately the vast majority of these cases are benign and need no labs, imaging or treatment. Patients should be advised not to wear contact lenses until symptoms resolve [11].

Pitfall | **Failure to identify the location of the blood can lead to confusion between a subconjunctival hemorrhage (seen laterally in the sclera) and a hyphema (seen in the lower portion of the iris), which have different treatments and follow-up needs**

Pre-septal versus septal cellulitis

Patients with pre-septal or septal (orbital) cellulitis will frequently present stating that they have "eye pain." Differentiation between these two processes is crucial as

septal cellulitis is potentially vision threatening. Pre-septal cellulitis is more commonly encountered in pediatric patients, though it does occur in adults as well. It involves the eyelid and anterior structures of the eye. It is more common and milder than orbital cellulitis, which affects the tissues inside the orbit. Orbital cellulitis can be vision and even life threatening. Both conditions can present with red swollen skin around the eye. The exam findings that raise concern for septal cellulitis are pain with extraocular movement, ophthalmoplegia, and proptosis. Frequently a history of recent bacterial rhinosinusitis is described. If the diagnosis is uncertain, computed tomography (CT) should be completed [12]. Orbital cellulitis should be treated with intravenous (IV) vancomycin and a cephalosporin. These patients should be referred to the ED for hospitalization. Pre-septal cellulitis may at times require hospitalization in patients under age one year, those who have failed outpatient antibiotics, or those who are toxic in appearance, but most patients can be treated as outpatients. Antibiotic coverage should include coverage for methicillin-resistant *Staphylococcus aureus* (MRSA) with trimethoprim-sulfamethoxazole or clindamycin, plus a beta-lactam such as amoxicillin-clavulanate or a cephalosporin.

KEY FACT | **Assessment of extraocular movements and evaluation for ophthalmoplegia and proptosis is essential in evaluating patients for orbital cellulitis. Failure to identify orbital cellulitis can be vision and life threatening.**

Throat

Sore throat is an extremely common complaint and has a broad differential diagnosis, ranging from viral illness to life-threatening conditions such as epiglottis and retropharyngeal abscess. In the majority of cases these conditions can be differentiated by history and physical examination.

KEY FACT | **Difficulty breathing, inability to swallow secretions, resistance/inability to lie flat, painful neck range of motion, and/or head held forward in the sniffing position are all indications of more serious pathology and warrant further investigation.**

Bacterial versus viral pharyngitis

Concern for "strep throat" brings many people to acute care centers. Group A *Streptococcus* (GAS), however, accounts for only 5–15% of pharyngitis in adults and 15–35% in children [13]. Other common bacterial causes of acute pharyngitis include gonorrhea, diphtheria, and *Fusobacterium*. Viruses account for the majority of cases of pharyngitis.

The diagnosis of GAS involves a combination of clinical decision tools and testing. Patients with GAS pharyngitis are generally between 3 and 44 years old; have a fever, tonsillar exudates, and anterior cervical lymphadenopathy; and are without a cough. The modified Centor criteria are used to guide testing and treatment (Table 1.2) [14].

KEY FACT | **Viruses account for the majority of cases of pharyngitis. Group A *Streptococcus* is responsible for up to 15% of cases in adults and 35% of cases in children.**

Polymerase chain reaction (PCR) testing and culture are often impractical in an urgent care practice given that it can take several days to get results. Alternatively, a point-of-care rapid antigen detection test (RADT) can be used. RADTs are approximately 85% sensitive and 96% specific, although there is significant variability of these values in different investigations [13]. A positive test result is helpful, but a negative result does not rule out the disease. To perform this test, vigorously swab both tonsils and the posterior pharynx. Obtaining an adequate sample is crucial, as sensitivities correlate directly to inoculum size [14].

Treatment

Treatment of GAS pharyngitis with antibiotics can reduce the duration of illness by 1–2 days, decrease the

Table 1.2 Modified Centor criteria.

Category	Criteria	Points
Age	3–14 years	1
	15–44 years	0
	≥45 years	–1
Tonsillar exudates	Present	1
Tender/swollen anterior cervical lymph nodes	Present	1
Temp >38 °C (100.4 °F)	Present	1
Cough	Absent	1

duration of communicability, and reduce the risk of suppurative complications (peritonsillar abscess, sinusitis, retropharyngeal abscess) and rheumatic fever. Antibiotics will not reduce the risk of acute glomerulonephritis [15].

Overprescribing of antibiotics has both personal (*Clostridium difficile*) and population (antibiotic resistance) risks. In the United States antimicrobial resistance leads to 48,000 deaths annually, underscoring the need to follow treatment guidelines when deciding to prescribe antibiotics for pharyngitis [16]. The decision as to whether or not to prescribe antibiotics can be guided by the patient's modified Centor criteria score (Table 1.3). Antibiotic choices are listed in Table 1.4.

KEY FACT | **The diagnosis of group A strep pharyngitis is made through a combination of clinical suspicion, clinical decision tools, and testing.**

Table 1.3 Application of modified Centor criteria [17, 18].

Score	Probability of GAS (%)	Recommendation
0–1	<10	No further testing; no antibiotics
2	11–17	RADT and/or culture; antibiotics only if positive
3	28–35	RADT and/or culture; antibiotics only if positive
4–5	51–53	RADT and/or culture; antibiotics if positive or if no testing is available

GAS, Group A *Streptococcus*; RADT, rapid antigen detection test.

Table 1.4 Treatment choices for Group A strep pharyngitis.

Antibiotic	Dose	Duration
Benzathine G penicillin	1.2 million units IM once If ≤27 kg: 600 K units IM	Once
PenVK	500 mg PO q 12 h If ≤27 kg: 250 mg PO q 12 h	10 days
If penicillin allergic (choose one)		
Clindamycin	300 mg PO q 8 h Peds: 7 mg/kg (max 300 mg) q 8 h	10 days
Cephalexin	500 mg PO q 12 h Peds: 20 mg/kg (max 500 mg) q 12 h	10 days

IM, intramuscularly; Peds, pediatrics; PO, orally; q, every.

Adjunctive treatment for pharyngitis (bacterial and viral) includes hydration, fever control, and, in most cases, corticosteroids. Corticosteroids improve severe throat pain and shorten the duration of symptoms [19]. Dexamethasone, 10 mg IV or orally in adults (0.6 mg/kg, max 10 mg in children), can be given as a one-time dose. Acetaminophen and/or NSAIDs may be used for fever and analgesia.

KEY FACT | **Corticosteroids improve severe throat pain and shorten the duration of symptoms for most bacterial and viral pharyngitis.**

Mononucleosis

Mononucleosis deserves special consideration for any patient with pharyngitis, particularly if antibiotics may be prescribed. Mononucleosis is a viral illness that is most commonly caused by Epstein–Barr virus or cytomegalovirus. The symptoms are generally malaise, fatigue, severe pharyngitis with posterior cervical lymphadenopathy, and fever.

Testing for mononucleosis can be complicated. A blood smear with >50% lymphocytes or >10% atypical lymphocytes can be used if other options are not available. This has an 87% positive predictive value and 93% negative predictive value [20]. Optimally, Epstein–Barr antibodies and nucleic antigen or an Epstein–Barr PCR should be checked. The heterophile antibody test, commonly referred to as a monospot, should not be used. It has poor sensitivity, especially within the first two weeks of symptoms, and can remain positive for up to a year after acute infection.

Treatment of mononucleosis is supportive and includes fluids, rest, analgesia, and fever control. Antibiotics are not indicated and, if inappropriately prescribed, can lead to the development of a macular erythematous, generalized rash. Steroids may be administered if there is concern for airway obstruction; otherwise, no significant benefit is conveyed by their use [21]. Patients should be advised to avoid contact sports for four to six weeks or until they are cleared by their primary care provider because of the risk of splenic rupture.

Gonococcal pharyngitis

Gonococcal pharyngitis must be considered in all sexually active patients, especially those who engage in oral sex. Patients may also have other sexually transmitted infection symptoms such as vaginal or urethral

discharge. Nucleic acid amplification testing (NAAT) can be done on oropharyngeal swabs [22, 23]. NAAT is better than culture and is the preferred diagnostic test for gonococcal pharyngitis [24]. The decision to treat versus wait for confirmatory testing is based on the clinical suspicion. Treatment is ceftriaxone 500 mg intramuscularly (IM) once for patients <150 kg and 1 g IM for patients ≥150 kg [25].

Peritonsillar abscess

Peritonsillar abscesses (PTAs) are the most common cause of deep neck space infections [26]. PTAs form next to the palatine tonsils and are generally preceded by pharyngitis/tonsillitis. The cause is usually polymicrobial, the predominant organisms being GAS, *Staphylococcus aureus* (including MRSA), and respiratory anaerobes. Patients typically present with a sore throat, voice change, and trismus. On examination there is unilateral tonsillar enlargement, often with exudate, and contralateral displacement of the uvula. Rarely, bilateral PTAs do occur, making diagnosis challenging. Although uncommon, a PTA can lead to life-threatening airway obstruction.

Imaging is not required to diagnose a PTA, although it may be necessary if there is diagnostic uncertainty. A CT scan with IV contrast or an ultrasound is the modality of choice. Ultrasound has the advantage of avoiding radiation exposure. It can also be used for live guidance if drainage is necessary.

Although it was previously believed that all PTAs require drainage, that has now been brought into question [27, 28]. Hemodynamically stable, well-appearing patients without concern for airway compromise may be trialed with IV antibiotics, IV steroids, and IV fluids, then discharged with a course of oral antibiotics.

Either clindamycin 900 mg, ampicillin-sulbactam 3 g, or piperacillin-tazobactam 4.5 g can be used as a first-dose IV agent. For patients with severe infections, vancomycin can be added, particularly if MRSA in the area has a high resistance to clindamycin. Dexamethasone 10 mg IV can help to reduce pain and swelling. Supportive care includes hydration, fever control, and analgesia.

Stable, well-appearing patients with PTA who are able to tolerate oral fluids can be discharged with a course of amoxicillin-clavulanate 875 mg orally twice a day or clindamycin 300 mg every 8 hours for 14 days.

Retropharyngeal abscess, epiglottitis, and Ludwig angina

Retropharyngeal abscess, epiglottitis, and Ludwig angina are deadly deep neck space infections that must be considered in patients with severe throat pain. Retropharyngeal abscess is much more common in children, but it can occur in adults. Epiglottitis was previously primarily a disease of children, but since the introduction of the *Haemophilus influenza* type b vaccine the most common patient now is an adult. Patients with Ludwig angina typically have difficulty swallowing because their tongue is displaced due to swelling of the floor of the mouth. Patients with these illnesses are usually ill appearing and may exhibit concerning symptoms such as dyspnea, inability to swallow secretions, resistance/inability to lay flat, painful neck movement, and/or head being held forward in the sniffing position. The presence of any of these findings portends a more serious infection. These diagnoses should also be considered when the severity of the throat pain does not correlate with the physical examination or if stridor is auscultated.

All of these conditions require emergent care due to a potentially unstable airway. Patients should be transported to the hospital by ambulance. While awaiting transport, leave the patient in a position of comfort and obtain IV access in adults. Consider deferring IV access in children due to the risk of respiratory distress induced by crying. The diagnosis is confirmed via CT scan (if the patient is stable enough to get the study) or in the operating room by direct visualization. Treatment involves parenteral antibiotics, steroids, surgical consultation, and admission.

KEY FACT | **In the past epiglottitis was encountered more frequently in children, but adults have become the primary group with this disease.**

Ear

Acute otitis media

Acute otitis media (AOM) is an infection of the middle ear that typically follows an upper respiratory infection. Patients are usually children who present with fever and ear pain or ear pulling. They may have decreased hearing on the affected side.

The patient will likely be febrile and otoscopy will show a moderately to severely bulging, erythematous, possibly opaque tympanic membrane (TM). If the patient complains of ear discharge, then a perforation may be seen. Pneumotoscopy will cause little or no movement of the TM.

AOM is difficult to differentiate from otitis media with effusion (OME). Both will present with ear pain and fullness, as well as a nonmobile TM, but OME is not infectious so patients will not exhibit signs of infection, such as fever or an erythematous TM. OME does not need treatment as it is a self-limiting disease.

All AOM patients should receive symptom control. Acetaminophen and/or ibuprofen can be used for analgesia and for fever control. For children older than three years, three drops of aqueous lidocaine 2% can be instilled into the external auditory canal once, as long as there is no concern for TM perforation [29].

The major decision in the treatment of AOM is whether or not to prescribe antibiotics immediately versus allowing for a period of observation. A delayed prescription for antibiotics is often referred to as a "wait and see prescription" (WASP). This involves providing a prescription for the patient but asking that it only be filled after 48–72 hours if symptoms persist [30]. Treatment versus WASP guidelines is listed in Table 1.5 and antibiotic treatment options in Table 1.6.

Mastoiditis

Mastoiditis is a suppurative complication of AOM where the infection spreads into the mastoid air cells posterior to the ear. Patients present with signs and symptoms of AOM plus pain *behind* the ear. On examination the ear is typically abducted away from the skull, purulent

Table 1.5 Treatment considerations for acute otitis media.

Immediate antibiotics	Consider WASP
<6 months old	6–23 months of age with unilateral otitis media and without severe symptoms[a]
6–23 months with bilateral AOM	Age ≥24 months with mild ear pain (unilateral or bilateral) and without severe symptoms[a]
≥6 months old with severe symptoms[a]	

AOM, acute otitis media; WASP, wait and see prescription.
[a] Severe symptoms are defined as moderate–severe ear pain, pain >48 hours, fever ≥39 °C [30].

Table 1.6 Treatment options for acute otitis media.

Antibiotic	Population	Dose	Duration
Amoxicillin	Children • New infection • Recurrence >15 d since last antibiotic	45 mg/kg/dose PO q 12 h (max 1 g/dose)	Age <2 yr: 10 d Age 2–5 yr: 7 d Age >5 yr: 5 d All ages with severe symptoms: 10 d
Amoxicillin-clavulanate	Adults	875 mg/125 mg PO q 12 h	5–7 d
Cefdinir	Mildly penicillin-allergic child	7 mg/kg/dose PO q 12 h (max 300 mg/dose)	5–10 d
Cefdinir	Mildly penicillin-allergic adult	600 mg PO q 24 h	5–10 d
Azithromycin	Severely penicillin-allergic child	10 mg/kg PO (max 500 mg) qD × 1 d then 5 mg/kg PO (max 250 mg) qD on days 2–5	5 d
Azithromycin	Severely penicillin-allergic adult	500 mg PO qD × 1 d then 250 mg PO qD days 2–5	5 d
Amoxicillin-clavulanate	Immunosuppressed, recently hospitalized, antibiotics within 30 d, failed prior treatment, child	45 mg/kg PO q 12 h (max 1 g/dose)	10 d
Ceftriaxone	Recurrent infection <15 days, child	50 mg/kg IV q 24 h (max 1 g/dose)	3 d

IV, intravenously; PO, orally; q, every; qD, daily.

otorrhea may be present, and the area behind the ear is erythematous and tender to palpation [31].

Any patient with concerns for AOM complications, such as mastoiditis or intracranial extension, should be referred to the ED for IV antibiotics and imaging.

KEY FACT | **Patients with acute otitis media should *not* have cranial nerve abnormalities, mastoid tenderness, vertigo, nystagmus, ataxia, or meningismus. If these are present, the patient needs prompt referral to an emergency department for further care.**

Tympanic membrane perforations

TM perforations can be due to AOM, blunt or penetrating trauma, barotrauma, blast injuries, or FBs. If the perforation is due to AOM then treat as per the AOM recommendations. If the perforation is from a retained FB, emergent referral to an ENT physician for removal is needed. Otherwise, most spontaneous TM perforations will heal spontaneously and do not require intervention.

If TM perforation is seen or suspected, do not irrigate the ear or perform pneumotoscopy. If the history provided is not consistent with a mechanism of TM perforation, then abuse should be suspected.

In the absence of infection, treatment focuses on pain management and keeping water out of the ear. This can be accomplished by placing a cotton ball coated with petroleum jelly into the external ear canal before showering or other exposure to water.

KEY FACT | **Tympanic membrane perforation associated with severe hearing loss, vomiting, ataxia, nystagmus, vertigo, facial nerve abnormality, Battle sign, or racoon eyes requires emergent ENT evaluation.**

Nose

Epistaxis

Epistaxis is a common patient presentation in the urgent care setting. It occurs more frequently in the winter months because of low humidity levels. Digital trauma, chemical irritants, and infections are common causes [32]. Most cases of epistaxis are self-limited but severe bleeds can be life threatening. Bleeds emanate from two primary locations. Anterior bleeds (90%) occur because of bleeding from the anterior nasal septum, and posterior bleeds (10%) occur from vessels in the posterior nasopharynx. Due to the noncompressible location of these posterior vessels, posterior bleeds tend to be high volume and are much more dangerous than their anterior counterparts.

The first step in managing nonmassive epistaxis is to compress the bleeding vessels. This is accomplished by the provider or patient squeezing the soft tissue portion of the nose, inferior to the nasal bone, for 5–10 minutes without interruption. Effective compression is achieved when the patient's voice sounds different ("nasal" voice) because of decreased airflow through the nose. After compression has been applied, the posterior oropharynx must be examined to assess for any ongoing bleeding down the back of the throat. A clot may be present, but the presence of active bleeding may indicate a posterior bleed. Posterior bleeds require transportation to an ED.

Applying ice to the palate can reduce blood flow to the nasal mucosa and help achieve hemostasis [33]. Applying ice to the exterior nose or the neck is ineffective [34].

If hemostasis is not achieved by compression, then a topical vasoconstrictor and/or hemostatic agent should be applied to the nasal mucosa with an atomizer or by soaking a gauze/pledget in the desired solution. Vasoconstrictors include oxymetazoline 0.05%, phenylephrine 0.5%, and epinephrine 1 mg/ml solution. Tranexamic acid is a hemostatic agent that can also be atomized or applied via soaked gauze/pledget to the mucosa to reduce bleeding [35]. Consider mixing a topical anesthetic with these agents for patient comfort should additional interventions be needed. Lidocaine with epinephrine atomized or applied topically works as both a vasoconstrictor and an anesthetic.

After the topical medications are applied and/or the gauze/pledget is removed, examine the nasal mucosa to see if hemostasis is achieved or, if not, if the site of the bleeding can be visualized. If hemostasis is not achieved and the site of bleeding can be identified, cautery with silver nitrate can be attempted. This must be done under direct visualization. Circumferential cautery around the bleeding site prior to cauterizing the vessel itself is often effective [32]. If the bleeding is bilateral, do not cauterize both sides of the septum due to the risk of causing a septal perforation.

If the bleeding site cannot be seen or if bleeding continues despite these measures, the next step is to place a nasal packing. Again, the posterior oropharynx should be assessed during and after each of these steps. If there

is concern for a posterior bleed, then transport the patient to the ED.

Anterior packing can be absorbable or nonabsorbable. Absorbable packing is usually hemostatic material that will dissolve over several days. Both are effective and the choice of packing is usually determined by what is readily available. Absorbable packing is preferred in patients who are at increased risk for rebleeding when the packing is removed [36]. This included patients with bleeding disorders and those on anticoagulant or antiplatelet medications. Packing should be placed parallel to the bottom of the nasal cavity and must be secured to the face to avoid posterior displacement, which can lead to aspiration and airway occlusion.

If the patient is hypertensive and hemostasis can be achieved, there is no indication for acute lowering of the blood pressure with IV medications. Rather, gradual lowering can be achieved with oral agents. Routine labs and imaging are not necessary for uncomplicated epistaxis. If a patient is on an anticoagulant with a measurable level, checking the level can be considered.

Patients in whom bleeding is controlled and are otherwise stable can be discharged. If a nonabsorbable nasal packing is in place, then follow-up should be arranged to have it discontinued in three days. Antibiotics are not necessary if the packing will be removed within 72 hours [37]. Absorbable nasal packing will disintegrate on its own and antibiotics are not necessary. All discharged patients should be advised to avoid nose blowing to avoid disrupting a hemostatic clot.

KEY FACT | **All patients with posterior bleeds (bleeding down the posterior oropharynx despite nasal soft tissue compression) are at risk for high-volume blood loss and must be referred to an ED.**

Foreign bodies

FBs of the ears and nose will bring many children, and a few adults, to urgent care centers. The typical presentation of a nasal FB is a child with unilateral nasal discharge without other upper respiratory symptoms. The typical presentation of an ear FB is a child presenting with unilateral ear pain without other signs of illness. Alternatively, a patient may present with a caregiver who saw the patient insert an FB or visualized the FB in the ear or nose.

General principles of FB treatment are as follows. If there is any concern for airway compromise (stridor, hypoxia, or respiratory distress) the patient requires immediate ambulance transport to an ED. Regardless of location, button batteries, live objects, and sharp FBs must be removed as quickly as possible. Button batteries can cause tissue necrosis within 15 minutes of exposure [38]. Uncomplicated FBs should not cause systemic illness or focal neurologic findings, including facial nerve palsies or cerebellar signs. When one FB is found, be sure to check the other head orifices for additional objects.

Complications of ear FBs include TM perforation with possible ossicle disruption leading to vertigo, ataxia and nystagmus, or otitis media. Complications of nasal FBs include aspiration, epistaxis, periorbital cellulitis, and meningitis. If any of the listed complications are present, the patient requires referral to an ED.

There are several removal techniques for nose and ear FBs. Before attempting removal, consider applying topical local anesthetic to improve patient comfort. For nose FBs, consider applying a topical vasoconstrictor (phenylephrine or oxymetazoline) to reduce swelling and facilitate removal. Live FBs should be killed prior to attempting removal. This can be done by instilling mineral oil, nonviscous lidocaine, or ethanol onto the object [39]. Ticks should be submerged in acetone, ethanol, or isopropyl alcohol [40]. Patients should be positioned during removal attempts such that if an object becomes dislodged it will fall out of the orifice.

Removal with alligator forceps is best when the FB can be easily visualized, does not have a smooth surface, and is nonfriable. Friable FBs may break into pieces when grasped. An alternative technique involves the use of topical adhesive. Place a small amount of topical adhesive onto the nonpadded end of a swab then bring that swab into contact with the FB for 30–60 seconds. The goal is then to remove the swab and the FB together. With this technique, the operator must take care to not come in contact with anything other than the FB. Another technique involves using a suction catheter with a small end (e.g. Frazier tip) to directly suck out the FB. This works best for smooth, round FBs. With this technique the operator must use low, continuous suction and be careful not to push the FB further into the orifice.

For nasal FBs, an initial attempt can be made at removal using a "positive pressure technique." This is done by applying positive pressure through the mouth

and having the only route of exit for the pressure be through the affected nostril. So if the FB is in the right nare, positive pressure is applied through the mouth while the left nostril is occluded. Positive pressure can be applied by the caregiver by using their mouth to puff air into the patient (sometimes referred to as the "parent's kiss") or through the use of a bag valve mask over the patient's mouth only.

For ear FBs, irrigation can also be attempted. This technique should not be used if the FB will expand when wet or if it is a button battery. Attempt to pass an angiocath past the FB then gently irrigate with body-temperature water.

Magnets can be used to remove metallic FBs, including magnets. A strong magnet (such as a pacemaker magnet) is applied to the end of a metallic instrument (e.g. hemostat or forceps) and then the instrument is brought into contact with the FB. The goal is to have the FB stick to the end of the instrument so that the two can be removed together.

Pearls for improving patient outcomes

- Patients with corneal abrasions, ulcers, and removed corneal foreign bodies are all treated with antibiotics and should see an ophthalmologist within 24 hours.
- Contact lens wearers with corneal abrasions or patients with corneal ulcers (regardless of contact lens use) require antipseudomonal antibiotic coverage and should be advised to refrain from wearing their contact lenses until they are cleared to do so by an ophthalmologist.
- Red flag warning signs for patients who present with a sore throat include difficulty breathing, inability to swallow secretions, resistance/inability to lie flat, painful neck range of motion, and/or head held forward in the sniffing position.
- The decision to prescribe antibiotics for possible streptococcal pharyngitis should be based on clinical suspicion, the modified Centor score, and testing.
- The need to prescribe antibiotics immediately versus providing a delayed prescription for acute otitis media is based on the patient's age and symptoms.
- Patients with persistent epistaxis or ongoing bleeding in the posterior oropharynx from epistaxis require immediate transport to an emergency department.
- Button batteries and sharp object foreign bodies require emergent removal.

References

1 Narayana S, McGee S. Bedside diagnosis of the 'Red Eye': a systematic review. *Am J Med* 2015; **128**(11): 1220–1224.e1.

2 Hogrefe C, Finnegan M, Herbert M. Keratitis and corneal ulcers. In: Johnson W, Nordt S, Mattu A, *et al*. (eds.), *CorePendium*. Burbank (CA): CorePendium, LLC; 2022. https://www.emrap.org/corependium/chapter/recdLUKfb1cY8ZJak/Keratitis-and-Corneal-Ulcers. Accessed February 2, 2024.

3 Leetch A, Howe M. Eye foreign bodies. In: Johnson W, Nordt S, Mattu A, *et al*. (eds.), *CorePendium*. Burbank (CA): CorePendium, LLC; 2022. https://www.emrap.org/corependium/chapter/recqNoTovkrodCLOa/Eye-Foreign-Bodies. Accessed February 2, 2024.

4 Bagheri N, Wajda B, Calvo C, *et al*. (eds.). Trauma. In: *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*. Philadelphia (PA), Lippincott Williams & Wilkins; 2020: pp. 49–121.

5 Lemon C. Conjunctivitis. In: Johnson W, Nordt S, Mattu A, *et al*. (eds.), *CorePendium*. Burbank (CA): CorePendium, LLC; 2023. https://www.emrap.org/corependium/chapter/recRN7jtAA3rtdyW7/Conjunctivitis. Accessed February 2, 2024.

6 Bagheri N, Wajda B, Calvo C, *et al*. (eds.). Conjunctiva/sclera/iris/external. In: *The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*. Philadelphia (PA), Lippincott Williams & Wilkins; 2020: pp. 216–264.

7 Herbert M, Swadron S. Corneal abrasion. In: Johnson W, Nordt S, Mattu A, *et al*. (eds.), *CorePendium*. Burbank (CA): CorePendium, LLC; 2022. https://www.emrap.org/corependium/chapter/recLlTOMumQUqII2r/Corneal-Abrasion. Accessed February 2, 2024.

8 Shipman S, Painter K, Keuchel M, *et al*. Short-term topical Tetracaine is highly efficacious for the treatment of pain caused by corneal abrasions: a double-blind, randomized clinical trial. *Ann Emerg Med* 2021; **77**(3): 338–344.

9 Arbour JD, Brunette I, Boisjoly HM, *et al*. Should we patch corneal erosions? *Arch Ophthalmol* 1997; **115**: 313–317.

10 Azari AA, Barney NP. Conjunctivitis: a systematic review of diagnosis and treatment. *JAMA* 2013; **310**(16): 1721–1729.

11 Mimura T, Usui T, Yamagami S, *et al*. Recent causes of subconjunctival hemorrhage. *Ophthalmologica* 2010; **224**(3): 133–137.

12 Botting AM, McIntosh D, Mahadevan M. Paediatric pre- and post-septal peri-orbital infections are different diseases. A retrospective review of 262 cases. *Int J Pediatr Otorhinolaryngol* 2008; **72**(3): 377–383.

13 Mustafa Z, Ghaffari M. Diagnostic methods, clinical guidelines, and antibiotic treatment for group a streptococcal

pharyngitis: a narrative review. *Front Cell Infect Microbiol* 2020; **10**: 563627.

14 Kurtz B, Kurtz M, Roe M, *et al*. Importance of inoculum size and sampling effect in rapid antigen detection for diagnosis of streptococcus pyogenes pharyngitis. *J Clin Microbiol* 2000; **38**: 279–281.

15 Van Brusselen D, Vlieghe E, Schelstraete P, *et al*. Streptococcal pharyngitis in children: to treat or not to treat? *Eur J Pediatr* 2014; **173**(10): 1275–1283.

16 Centers for Disease Control and Prevention (2022). About antibiotic resistance. https://www.cdc.gov/drugresistance/about.html. Accessed January 24, 2024.

17 Al-Baghli NA, Al Saif A, Al Dorazi SA, *et al*. Prevalence of positive rapid antigen detection test for group A *Streptococcus* among patients with respiratory symptoms in Eastern Province, Kingdom of Saudi Arabia. *Cureus* 2023; **15**(11): e48607.

18 Kalra MG, Higgins KE, Perez ED. Common questions about streptococcal pharyngitis. *Am Fam Physician* 2016; **94**(1): 24–31. Erratum in *Am Fam Physician* 2017;**95**(7):414.

19 Sadeghirad B, Siemieniuk RAC, Brignardello-Petersen R, *et al*. Corticosteroids for treatment of sore throat: systematic review and meta-analysis of randomised trials. *BMJ* 2017; **358**: j3887.

20 Shi T, Huang L, Luo L, *et al*. Diagnostic value of serological and molecular biological tests for infectious mononucleosis by EBV in different age stages and course of the disease. *J Med Virol* 2021; **93**(6): 3824–3834.

21 Rezk E, Nofal YH, Hamzeh A, *et al*. Steroids for symptom control in infectious mononucleosis. *Cochrane Database Syst Rev* 2015; **2015**(11): CD004402.

22 Workowski KA, Bolan GA. (2015). Sexually transmitted diseases treatment guidelines, 2015. *MMWR Recomm Rep*. **64**(RR-03):1–137. Erratum in *MMWR Recomm Rep*. 2015;**64**(33):924.

23 Cherabie JN, Liang SY, Reno H. Diagnostics for gonorrhea and chlamydia in the emergency department: fight smarter not harder. *Ann Emerg Med* 2021; **77**(4): 411–413.

24 Bachmann LH, Johnson RE, Cheng H, *et al*. Nucleic acid amplification tests for diagnosis of *Neisseria gonorrhoeae* oropharyngeal infections. *J Clin Microbiol* 2009; **47**(4): 902–907.

25 Centers for Disease Control and Prevention (2020). Update to CDC's treatment guidelines for gonococcal infection. https://www.cdc.gov/mmwr/volumes/69/wr/mm6950a6.htm. Accessed January 24, 2024.

26 Schraff S, McGinn JD, Derkay CS. Peritonsillar abscess in children: a 10-year review of diagnosis and management. *Int J Pediatr Otorhinolaryngol* 2001; **57**: 213–218.

27 Rezk E, Nofal YH, Hamzeh A, *et al*. Steroids for symptom control in infectious mononucleosis. *Cochrane Database Syst Rev* 2015; **2015**(11): CD004402.

28 Forner D, Curry DE, Hancock K, *et al*. Medical intervention alone vs surgical drainage for treatment of peritonsillar abscess: a systematic review and meta-analysis. *Otolaryngol Head Neck Surg* 2020; **163**(5): 915–922.

29 Prasad S, Ewigman B. Use anesthetic drops to relieve acute otitis media pain. *J Fam Pract* 2008; **57**(6): 370–373.

30 Lieberthal AS, Carroll AE, Chonmaitree T, *et al*. The diagnosis and management of acute otitis media. *Pediatrics* 2013; **131**(3): e964–e999. Erratum in *Pediatrics* 2014;**133**(2):346.

31 Panakos P, Jain R. Mastoiditis. In: Johnson W, Nordt S, Mattu A, *et al*. (eds.), *CorePendium*. Burbank (CA), CorePendium, LLC; 2023. https://www.emrap.org/corependium/chapter/recN1m7VRM86BVq03/Mastoiditis. Accessed January 28, 2024.

32 Krulewitz NA, Fix ML. Epistaxis. *Emerg Med Clin North Am* 2019; **37**(1): 29–39.

33 Porter M, Marais J, Tolley N. The effect of ice packs upon nasal mucosal blood flow. *Acta Otolaryngol* 1991; **111**: 1122–1125.

34 Teymoortash A, Sesterhenn A, Kress R, *et al*. Efficacy of ice packs in the management of epistaxis. *Clin Otolaryngol Allied Sci* 2003; **28**: 545–547.

35 Janapala RN, Tran QK, Patel J, *et al*. Efficacy of topical tranexamic acid in epistaxis: a systematic review and meta-analysis. *Am J Emerg Med* 2022; **51**: 169–175.

36 Tunkel DE, Anne S, Payne SC, *et al*. Clinical practice guideline: nosebleed (epistaxis). *Otolaryngol Head Neck Surg* 2020; **162**(1_suppl): S1–S38.

37 Biswas D, Mal RK. Are systemic prophylactic antibiotics indicated with anterior nasal packing for spontaneous epistaxis? *Acta Otolaryngol* 2009; **129**: 179–181.

38 Litovitz T, Whitaker N, Clark L, *et al*. Emerging battery-ingestion hazard: clinical implications. *Pediatrics* 2010; **125**(6): 1168–1177.

39 Leffler S, Cheney P, Tandberg D. Chemical immobilization and killing of intra-aural roaches: an in vitro comparative study. *Ann Emerg Med* 1993; **22**(12): 1795–1798.

40 Orobello NC, Dirain CO, Kaufman PE, *et al*. Efficacy of common reagents for killing ticks in the ear canal. *Laryngoscope Investig Otolaryngol* 2018; **3**(6): 492–495.

CHAPTER 2
Management of Genitourinary Complaints

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Introduction

Genitourinary complaints are common in most acute care centers. Fortunately, most of these complaints are straightforward and do not require much intervention. However, some cases exhibit mild presentations of more serious diseases that require a more thorough workup. The acute care provider must maintain a broad differential and refrain from misdiagnosing or mismanaging these more acute disease processes.

Pitfall | **Over-reliance on a negative urinalysis to rule out renal colic**

Nephrolithiasis is an increasingly encountered medical problem, accounting for nearly 600,000 emergency department visits, 2 million primary outpatient visits, and approximately $2.1 billion in healthcare-related expenditures in the United States per year according to a population study [1]. The prevalence continues to increase worldwide, with multiple countries reporting increases in both prevalence and incidence [2]. The peak incidence of nephrolithiasis occurs between the ages of 35 and 50. Men remain at the greatest risk, with a male-to-female ratio of approximately 3 : 1, although that gap appears to be narrowing with an increase in the number of female patients experiencing nephrolithiasis rising compared to men [3]. Common presenting symptoms include flank pain radiating down into the groin, visible distress, urinary frequency, and, sometimes, hematuria.

Historically, using the presence of hematuria on urinalysis to rule in/out a kidney stone has been taught. Unfortunately, this approach often leads to a misdiagnosis that can place the patient at risk for complications of a kidney stone, such as urinary obstruction, hydronephrosis, renal failure, and renal capsule rupture. Using the urinalysis as a screening tool has been shown to be insufficient in several studies. Urinalysis looking for the presence of hematuria with at least five red blood cells was associated with a sensitivity of 86% [4].

KEY FACT | **Hematuria, even microscopic, can be absent in approximately 14% of patients with a kidney or ureteral stone.**

A urinalysis remains useful in the workup of flank pain. It can be used to identify other possible causes of flank pain, such as pyelonephritis. Consequently, there can be a concomitant urinary tract infection associated with a kidney stone. These infections are more difficult to treat and often require hospitalization, parenteral antibiotics, and possible surgical intervention as inadequate antibiotic penetration occurs due to the obstruction. Immediate urologic consultation is recommended for these patients, especially if obstruction is present. Urinalysis also detects hematuria, which still warrants outpatient referral to a urologist if the workup yields no cause for the hematuria.

KEY FACT | **A urinary tract infection associated with a kidney stone is a urologic emergency.**

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Box 2.1 Alternative causes of acute flank pain

- Appendicitis
- Diverticulitis
- Testicular/ovarian torsion
- Pyelonephritis
- Perinephric hematoma
- Renal capsule rupture
- Abdominal aortic aneurysm
- Neoplasm (usually causing obstruction)

The sensitivity and specificity of computed tomography (CT) scan for the detection of kidney stones are 96% and 97%, respectively [5]. New technology in the realm of CT scanning has produced faster and more precise scanning machines. The limitations that once delayed a patient's stay in the acute care center have significantly decreased so that the diagnosis is now made or excluded in a relatively short period of time. CT scanning can also detect other significant causes of flank pain, even if hematuria is present. Some of these alternative causes can be found in Box 2.1. Hematuria can be present in a number of these pathologic processes; however, hematuria can be absent, leading to no further workup and significant morbidity if missed. CT scan without intravenous (IV) contrast has long been the standard imaging for patients with suspected ureteral colic. These studies are excellent for size and location of renal and ureteral stones. However, lack of contrast limits the imaging ability to diagnose other causes of pain and hematuria. More recent studies suggest that if other etiologies of pain are to be considered, IV contrast-enhanced CT can safely exclude obstructive ureterolithiasis [6]. Therefore, CT scanning either with or both with and without contrast is a valuable modality in the evaluation of any patient presenting with flank pain, with strong encouragement to utilize IV contrast if the diagnosis is in question. In some patients with very high pretest probability of ureteral colic and low risk for complication, ultrasound is encouraged to be the first-line imaging option, and in some cases the only option [7].

Pitfall | **Failure to consider Fournier gangrene as a cause of acute scrotal pain**

Fournier gangrene is a rapidly spreading, life-threatening infection of the superficial and deep perineal tissues.

Box 2.2 Treatment of Fournier gangrene

Vancomycin + clindamycin OR linezolid
PLUS
Piperacillin/tazobactam
OR
Carbapenem

The mortality rate remains high at 20% [8]. Patients at risk for Fournier gangrene are more frequently men with comorbid diseases. Diabetes, immunocompromise, renal failure, hypertension, and heart, liver, lung, and kidney diseases all increase mortality [9].

The classic presentation is like those of other necrotizing infections: extreme pain, "soap water" discharge, and crepitus on exam of a swollen, erythematous perineum. However, some authors suggest that many of these patients may be presenting earlier, without a clear and obvious sign of significant infection [10]. A high index of suspicion for necrotizing infection especially in at-risk individuals can aid in identification and early referral to surgical centers to help diminish the mortality even further in these patients. Although surgical debridement is the mainstay of treatment for necrotizing infections, early administration of antibiotics is paramount, especially in cases of sepsis. In necrotizing infections such as Fournier gangrene (Box 2.2), the polymicrobial nature of the infection requires broad-spectrum coverage of gram-positive, gram-negative, and anaerobic bacteria. In addition, toxin inhibition is required, typically with clindamycin. Newer studies provide further evidence that linezolid in place of vancomycin and clindamycin is equivalent, with a need for a smaller number of antibiotics [11].

Pitfall | **Failure to recognize and appropriately treat complicated urinary tract infections**

Urinary tract infections (UTIs) are common occurrences in the acute care center, accounting for almost 8 million outpatient visits each year [12]. Women are most affected because of their shorter urethra and its proximity to the vagina and the anus. As a result, one-third of women in the United States will experience a UTI by the time they are 25 years old and one-half will experience one by 35 years old [13]. UTI not only afflicts our working population but also the old and the very young. In the elderly population it is the second most

common infectious cause for hospital admission and was the highest paid Medicare reimbursement in 1998 [14]. In the pediatric population the incidence is up to 5% of febrile illnesses presenting to a physician. Overall, this very common disease correlates with high healthcare costs as patients frequently present and are invariably placed on some antibiotic regimen.

Once the diagnosis of a UTI is established, the acute care provider must differentiate between a simple and a complicated infection. This has nothing to do with the severity of symptomology or with significantly abnormal laboratory values. Rather, it describes a patient population that has either a significant comorbidity or a structural or anatomical abnormality of their urogenital system that predisposes them to an increased risk of worsening and/or significant complications. Complications can include urosepsis, acute renal insufficiency, and perinephric abscess. A list of these risk factors is given in Box 2.3. Any patient with these risk factors who is diagnosed with a UTI needs to be managed as if they have a complicated UTI.

Diabetes mellitus deserves special mention because people with diabetes are at the highest risk for complications surrounding inadequately treated UTIs. Glucosuria predisposes those with diabetes to asymptomatic bacteriuria, which usually results in ascension up the urinary tract leading to a UTI. Because of decreased renal perfusion and decreased immune response, infections with atypical pathogens such as *Pseudomonas* and fungi can occur, and inadequate antimicrobial coverage often results in serious complications. Perinephric abscesses and emphysematous pyelonephritis are complications seen almost exclusively in those with diabetes; the latter carries a mortality of 25% with likely loss of the affected kidney [15].

KEY FACT | **Emphysematous pyelonephritis is a complication almost exclusively found in people with diabetes and carries a mortality of 25%.**

Complicated UTIs require longer regimens of antimicrobial therapy that will perfuse the urinary tract as well as the parenchyma of the organs involved. The approach to treatment of a complicated UTI is to start a broad-spectrum antibiotic after obtaining a urine culture. Treatment regimens can be found in Box 2.4. Fluoroquinolones have been first-line treatment because they maintain heavy concentration in the urine and renal parenchyma. Unless contraindicated (i.e. pregnancy, pediatrics), it is recommended to start with a fluoroquinolone, follow up with the culture results, and adjust therapy accordingly. If local resistance of community uropathogens exceeds 10% resistance to fluroquinolones, alternate therapy should be considered, such as trimethoprim-sulfamethoxazole [16]. If sensitivities are not known, the Infectious Diseases Society of America (IDSA) recommends a one-time parenteral dose of long-acting agent, such as ceftriaxone. Current guidelines caution over the use of oral cephalosporins, due to possible increased recurrence rates [16].

Box 2.3 Risk factors for complicated urinary tract infection

- Diabetes mellitus
- Male sex
- Neonates
- Elderly
- Recent hospitalization
- Indwelling catheters
- Genitourinary surgery (i.e. nephrostomy, stents)
- Renal insufficiency
- Vesicoureteral reflux
- Urethral valves
- Pregnancy
- Immunocompromise
- Concomitant nephrolithiasis

Box 2.4 Treatment regimens for complicated urinary tract infection in adults

Outpatient:

- Ciprofloxacin 500 mg twice a day or levofloxacin 750 mg once daily
- Trimethoprim-sulfamethoxazole 1 double-strength tablet twice daily
- Third-generation cephalosporin such as ceftibuten, cefdinir, or cefpodoxime

Inpatient:

- Ciprofloxacin 400 mg intravenously (IV) twice a day or levofloxacin 750 mg IV once daily
- Third-generation cephalosporin such as ceftriaxone, ceftazidime, or cefotaxime
- Imipenem/cilastatin
- Piperacillin/tazobactam

Newer literature suggests that oral cephalosporin treatment for these patients may be safe if first-line agents are contraindicated or resistant [17]. Disposition is dependent on the patient's presentation, but requires a low threshold to hospitalize these patients for at least a day of parenteral antibiotics. If they are discharged home, they should have very close follow-up (i.e. within 48 hours) to evaluate response to therapy and to amend therapy based on culture results.

Pitfall | **Failure to accurately diagnose and manage acute scrotal pain**

Acute scrotal pain is an infrequent complaint in the acute care setting. However, a patient who presents with this complaint should have immediate evaluation by the acute care provider to determine the possible causes. Studies have shown that it is difficult to differentiate testicular torsion from torsion of the appendix testis and epididymitis/orchitis based on presenting symptoms and historical features alone [18].

Testicular torsion is one of the most common surgical emergencies occurring in males, with an annual incidence of 1 in 4000 among patients younger than 25 years, with a bimodal distribution, the largest peak occurring around puberty (accounting for 65% of all torsions) and another smaller peak in the first year of life [19, 20]. A delay in diagnosis risks the viability of the affected testis. Epididymitis is the fifth most common urologic diagnosis in men between the ages of 18 and 50, with most cases occurring between 20 and 39 years [21]. Among sexually active men younger than 35 years, acute epididymitis is most frequently caused by sexually transmitted organisms, specifically *Chlamydia trachomatis* and *Neisseria gonorrhoeae*. In contrast, the most common pathogens isolated in infectious epididymitis in men >35 years are coliform bacteria, with *Escherichia coli* accounting for most cases [22, 23].

Differentiating between these two etiologies for acute scrotal pain is a diagnostic dilemma for the acute care provider. While some studies suggest that patients with testicular torsion tend to present earlier than those with other diagnoses, this historical element should not be used to distinguish between these entities alone. Furthermore, the classic pattern of acute-onset unilateral testicular pain may not be present in all cases. In a retrospective review of patients with testicular torsion, 89% of patients presented with this symptom. The investigators also found associated groin, abdominal, or thigh pain in 34% of these patients, indicating that alternative areas of pain may represent the earliest or predominant symptom in testicular torsion [19]. The physical examination findings in patients who present with acute scrotal pain may also be misleading. Specifically, the absence of the cremasteric reflex (which is considered the most sensitive physical examination finding in testicular torsion) may be unreliable. In a study of 225 healthy boys, investigators noted that the cremasteric reflex was present in all boys older than 30 months but in less than half of those younger than 30 months [24]. Similarly, although rare, case reports exist of patients with testicular torsion and an intact cremasteric reflex [25]. Likewise, Prehn's sign, commonly used to differentiate torsion from epididymitis by relief of pain when the testicle inflamed by epididymitis is elevated, is not specific [26]. Other classic physical examination findings that suggest testicular torsion, including a higher lie of the affected testis, horizontal lie compared to the contralateral testis, and displacement of the epididymis from its usual posterior position, may not be present, and the absence of any one or more of these findings should not be used to rule out the diagnosis of torsion.

While no single historical or exam finding is diagnostic for testicular torsion, providers must consider the overall picture with all components. The Testicular Workup for Ischemia and Suspected Torsion (TWIST) is a validated scoring system that may help risk stratify patients (Box 2.5) [27, 28]. Patients with a score of zero are not likely to have a testicular torsion, some studies suggesting up to 100% negative predictive value [28]. Importantly, this should not dissuade a provider from obtaining a scrotal ultrasound, which can help differentiate other

Box 2.5 TWIST score

Testicular swelling	Yes, +2
Hard testes	Yes, +2
Absent cremasteric reflex	Yes, +1
Nausea or vomiting	Yes, +1
High-riding testicle	Yes, +1
	Risk:
	0 = low, 1–5 = intermediate, 6–7 = high

causes of an acute scrotum. A score of zero may however aid in determining the urgency of ultrasound if immediate ultrasound is not available at a presenting site.

KEY FACT | **In healthy children under 30 months old less than half had a cremasteric reflex, making it an unreliable physical exam finding when evaluating for torsion.**

Because of these diagnostic dilemmas and the potentially disastrous outcome if there is delay of diagnosis, it is critical for the acute care provider to consider testicular torsion in all cases of acute scrotal pain. When the diagnosis of testicular torsion is questionable, the provider must obtain Doppler ultrasonography and/or urologic consultation expeditiously. Only after the diagnosis of torsion has been excluded should other diagnoses be considered.

If epididymitis is the diagnosis, the patient must be managed appropriately. Only half of patients 18–35 years with a diagnosis of epididymitis receive appropriate treatment [29, 30]. Per Centers for Disease Control (CDC) recommendations, empiric therapy for epididymitis is indicated before laboratory results are available and should be guided by the patient's age and sexual history. Testing for *N. gonorrhoeae* and *C. trachomatis* should be performed in sexually active patients <35 years using the diagnostic modality readily available to the clinician (i.e. culture, nucleic acid hybridization test, or nucleic acid amplification test). Treatment regimens can be found in Box 2.6 [31]. Adjunctive treatment modalities, including bed rest, scrotal elevation, and analgesics, are also recommended until fever and inflammation have subsided [31]. Of equal importance to choosing the appropriate antibiotic regimen is thorough patient education. Patients who have acute epididymitis that is suspected or confirmed to be caused by *N. gonorrhoeae* or *C. trachomatis* should be instructed to refer recent sexual partners (within 60 days) for evaluation and treatment. In addition, they should be instructed to abstain from sexual intercourse until they and their partners have been adequately treated [31].

Box 2.6 Treatment regimens for acute epididymitis

For acute epididymitis most likely caused by gonococcal or chlamydial infection:
- ceftriaxone 500 mg intramuscularly (IM) in a single dose (if >150 kg, 1 g IM in a single dose) PLUS
- doxycycline 100 mg orally twice a day for 10 days

For acute epididymitis most likely caused by chlamydia, gonorrhea, or enteric organisms (men who practice insertive anal sex):
- ceftriaxone 500 mg IM in a single dose (if >150 kg, 1 g IM in a single dose) PLUS
- levofloxacin 500 mg orally once daily for 10 days

For acute epididymitis most likely caused by enteric organisms only or with negative confirmatory gonococcal testing:
- levofloxacin 500 mg orally once daily for 10 days

Pitfall | Failure to suspect pregnancy

Every woman of childbearing age should be presumed pregnant until proven otherwise. Multiple studies have shown that historical and physical exam features are inconsistent and unreliable in determining the possibility of pregnancy. Despite recording various historical variables—including the date of the patient's last menstrual period, whether the patient's menstrual period was on time, birth control usage, and whether the patient suspected she was pregnant—one study showed that there was still a 10% chance of pregnancy being overlooked [32]. Furthermore, women may not associate certain symptoms with early pregnancy. Additionally, some women may be unaware or, worse, deny being pregnant when they really are, termed a cryptic pregnancy. One population study shows that as many as 1 in 475 pregnancies is unknown at 20 weeks and 1 in 4500 pregnancies is unknown at delivery [33]. Another study showed that among fertile patients who presented to the emergency department for any reason, the prevalence of unrecognized pregnancy (defined as "pregnancy not definitely known to exist") in those with abdominal pain or pelvic complaints was found to be 13% [34].

Although patients may report results based on home pregnancy testing, acute care providers should maintain a fair amount of skepticism. The accuracy of home pregnancy tests relies on several factors, including the ability of patients to carefully follow the test kit instructions, the timing of testing (i.e. number of days beyond the missed menstrual period), and the ease with which test results can be interpreted.

Given the poor reliability of the patient's history in determining pregnancy status, and the potentially disastrous outcomes of missing the diagnosis of pregnancy (i.e. prescribing teratogenic studies and therapies), it is recommended that laboratory testing (urine or serum

HCG) be performed on all females of childbearing age to determine the possibility of pregnancy.

Pitfall | **Failure to treat asymptomatic bacteriuria in pregnancy**

Asymptomatic bacteriuria is a diagnosis of an infection of the genitourinary system where the patient does not experience any symptoms. In most patients it is an incidental finding on a urinalysis that is usually being ordered for another reason. The prevalence of asymptomatic bacteriuria in the young adult female population is fairly common, somewhere between 2 and 10%, and more prevalent in populations from lower socioeconomic status [35]. Failure to treat asymptomatic bacteriuria in most patient populations will typically result in no adverse events. In these cases, the infection will most commonly be cleaned out by the body's immune response or, rarely, will progress to a symptomatic infection, resulting in the patient seeking medical attention.

However, the pregnant population deserves special attention when it comes to asymptomatic bacteriuria. Pregnancy-associated physiologic changes place the patient at risk for ascension of the bacteria up the urinary tract, causing worsening infection. Prior literature showed that 30% of pregnant patients who have untreated asymptomatic bacteriuria go on to develop pyelonephritis [36]. There are also risks for the fetus. Pregnancies associated with untreated asymptomatic bacteriuria are at increased risk of preterm labor and delivery as well as low-birth-weight babies. For this reason, prenatal clinics screen for asymptomatic bacteriuria as primary prevention for the complications. However, patients of lower socioeconomic status are more likely to delay or never establish prenatal care and will often present to acute care centers for any complaints that may occur during the pregnancy. Fortunately, a common practice in most acute care centers is to confirm a pregnancy with a urine pregnancy test. It is recommended that the acute care provider also check a urinalysis in combination with the pregnancy test to screen for asymptomatic bacteriuria.

Due to the increase in screening, the incidence of pyelonephritis during pregnancy has significantly lowered such that the recommendations for screenings have changed [37, 38]. Patients with prenatal care do not require multiple screenings for asymptomatic bacteriuria nor multiple treatments [38]. Therefore, patients who have established prenatal care who have already had screening or may have already been treated previously for asymptomatic bacteriuria may not require routine urine testing. However, acute care providers who are seeing patients who have not yet established prenatal care or are unable to establish prenatal care should still perform urine screening. For any patient where screening is performed, urine culture should also be obtained to assess for antimicrobial resistance.

KEY FACT | **Historically, 30% of pregnant patients with untreated asymptomatic bacteriuria may go on to develop pyelonephritis.**

Contrary to the common practice in nonpregnant patients, the current recommendations are to treat asymptomatic bacteriuria in pregnant patients with antibiotics. Treatment has been shown to decrease the risk of pyelonephritis by 80% and the risk of low-birth-weight infants by 15% [36]. There has been an increase in *Escherichia coli* resistant to ampicillin, making the penicillins obsolete. Cephalosporins and nitrofurantoin are the most used antibiotics for the treatment of asymptomatic bacteriuria and are equally effective.

KEY FACT | **Treating asymptomatic bacteriuria in pregnancy decreases the risk of pyelonephritis by 80% and the risk of low-birth-weight infants by 15%.**

Pitfall | **Failure to consider ovarian torsion in females who present with acute lower abdominal pain**

Ovarian torsion is a frequently taught yet highly underrecognized cause of acute abdominal pain. It is the fifth most common gynecologic emergency, accounting for 2.7% of acute gynecologic complaints [39–41]. Early diagnosis and treatment are essential to salvage the affected ovary. Unfortunately, delay in diagnosis is more common, leading to a reported ovarian salvage rate of less than around 45% [42].

KEY FACT | **Due to delay in diagnosis of ovarian torsion, the rate of ovarian salvage once the diagnosis is made is less than 10%.**

The challenges to establishing an early diagnosis of ovarian torsion are multifactorial. Clinicians relying on the classic history and physical exam will find there are some shortcomings. Classically, patients with ovarian torsion present with abrupt-onset, colicky, unilateral lower abdominal pain that radiates to the flank or groin and progressively worsens over several hours, often associated with nausea and/or vomiting. In one study, however, this "classic" presentation was only found in 44% of patients with confirmed ovarian torsion [43]. Abrupt onset of pain is only seen in about 50% of patients confirmed to have ovarian torsion. Nausea and vomiting are seen in 70% of patients with torsion. The physical exam is equally unreliable as about 35% of patients will have no abdominal tenderness on exam nor adnexal tenderness on pelvic exam [41].

KEY FACT | **The "classic" presentation for ovarian torsion was found in only 44% of patients with confirmed ovarian torsion.**

Furthermore, the differential diagnosis for patients presenting with such complaints is broad and may include other emergency causes such as ectopic pregnancy, pelvic inflammatory disease, appendicitis, diverticulitis, ovarian cyst, and renal colic [39]. The frequent occurrence of these diseases may lead the clinician to overlook the less common diagnosis of ovarian torsion as the cause of the patient's acute abdominal pain. In fact, in a retrospective analysis of patients with surgically confirmed ovarian torsion, the diagnosis of ovarian torsion was considered in the admitting differential in only 47% of cases [43].

Though the diagnosis of ovarian torsion may be elusive based on history and physical examination alone, it is essential for the healthcare practitioner to consider it in the differential diagnosis of all female patients who present with acute lower abdominal pain. In cases when the diagnosis is likely, prompt sonographic evaluation and gynecologic consultation should be pursued.

Pitfall | **Failure to diagnose and treat pelvic inflammatory disease due to lack of classic symptoms**

Pelvic inflammatory disease (PID) is an infection of the upper genital tract in women. Most commonly caused by sexually transmitted infections (STIs), such as *N. gonorrhoeae* and *C. trachomatis*, PID is most prevalent in the young and sexually active population. The prevalence has been declining since the 1980s due to a heightened awareness in the general population and the increase in free STI clinics [44]. However, the rate of misdiagnosis remains high, leading to several complications including tubo-ovarian abscess, increased risk of ectopic pregnancy, and infertility.

The diagnosis of PID is very difficult due to variable presentation ranging from mild pelvic pain to peritonitis. The so-called classic presentation for PID of pelvic pain, vaginal discharge, and fever is frequently not found. There is not a single finding in the history, physical examination, or laboratory studies that is sensitive or specific for the diagnosis of PID. Even in the hands of experts, the history and physical examination were found to have a positive predictive value of only 65–90% [31, 44]. Furthermore, the presentation can often mimic other disease processes that may lead the acute care practitioner to pursue other diagnoses, such as appendicitis or a UTI.

KEY FACT | **The history and physical exam have a positive predictive value of 65–90% for the clinical diagnosis of pelvic inflammatory disease.**

In 2010, the CDC recognized the difficulty in making a diagnosis of PID. Unfortunately, its diagnostic criteria were sensitive but not specific for the diagnosis of PID. Subsequently, the CDC modified the diagnostic criteria in the 2015 Sexually Transmitted Infection Treatment Guidelines, and they were maintained in the guidelines published in 2021 [31]. They simplified the diagnostic criteria to lower the threshold to diagnose and treat. The diagnostic criteria can be found in Box 2.7 [31]. While each of these criteria alone still has poor specificity, the specificity increases as multiple criteria are met. If any of the minor criteria are met, further investigation and likely treatment are warranted.

In addition to these diagnostic criteria, the acute care provider must consider patient risk factors. Patients who are considered high risk are outlined in Box 2.8. Patients who are considered high risk who complain of abdominal or pelvic pain without an alternative etiology for their pain should be treated empirically for PID. Obtain laboratory studies as well, including swabs from the cervix for culture. However, because of the processing time of cervical cultures, antimicrobial therapy should be initiated in high-risk patients or patients with high suspicion prior to the results of the cultures returning.

Box 2.7 Diagnostic criteria for pelvic inflammatory disease

Minimum criteria
Presence of one or more of the following on pelvic examination:
- cervical motion tenderness
- adnexal tenderness
- uterine tenderness

Presence of the following in addition to the minimum criteria enhances the specificity for the diagnosis:
- fever >38.3 °C
- abnormal cervical mucopurulent discharge or cervical friability
- presence of abundant numbers of white blood cells on saline microscopy of vaginal fluid
- laboratory confirmed cervical infection with *C. trachomatis* or *N. gonorrhoeae*
- elevated erythrocyte sedimentation rate (ESR) and/or C-reactive protein (CRP)

Box 2.8 Risk factors for pelvic inflammatory disease

- Younger age of onset of sexual activity
- Multiple sexual partners
- Low socioeconomic status
- High community prevalence of gonorrhea or chlamydia
- Prior history of sexually transmitted infections
- Intercourse without barrier contraception
- Intercourse during menses

Treatment consists of antimicrobials directed against both *N. gonorrhoeae* and *C. trachomatis* as the clinical spectrum of either infection by these pathogens is similar. Recent serologic testing has determined that multiple other pathogens can also cause PID; however, the recommended antimicrobial regimen covers those pathogens as well. The recommended treatment algorithm changed in 2021. Treatment regimens can be found in Box 2.9 [31]. Modifications to the treatment regimen include an increase in the dosing of ceftriaxone due to increased resistance to cephalosporins by *N. gonorrhoeae*. New to the treatment regimen is the addition of metronidazole to the algorithm for concomitant treatment of bacterial vaginosis and anaerobes that cause upper genital tract infections. Hospitalization for parenteral antimicrobial administration is necessary in certain circumstances such as pregnancy, presence of a tubo-ovarian abscess, or inability to tolerate oral medications.

Box 2.9 Centers for Disease Control recommended treatment regimen for pelvic inflammatory disease

Outpatient:
- ceftriaxone 500 mg intramuscularly (IM) × 1 dose (if patient is >150 kg, give 1 g IM × 1 dose) PLUS
- doxycycline 100 mg orally (PO) twice a day (BID) for 14 days PLUS metronidazole 500 mg PO BID for 14 days

OR
- cefoxitin 2 g IM × 1 dose and probenecid 1 g PO × 1 dose PLUS
- doxycycline 100 mg PO BID for 14 days PLUS metronidazole 500 mg PO BID for 14 days

OR
- other parenteral third-generation cephalosporin (e.g. ceftizoxime or cefotaxime) PLUS
- doxycycline 100 mg PO BID for 14 days PLUS metronidazole 500 mg PO BID for 14 days

Inpatient:
- ceftriaxone 1 g IV every 24 h OR cefotetan 2 g IV every 12 h OR cefoxitin 2 g IV every 6 h PLUS
- doxycycline 100 mg IV or PO BID for 14 days PLUS metronidazole 500 mg PO BID for 14 days

Pearls for improving patient outcomes

- A urinalysis is inadequate when used as a screening tool for ruling in or out nephrolithiasis.
- Always consider Fournier gangrene, especially in at-risk patients with scrotal or perineal cellulitis.
- Always consider whether a urinary tract infection is complicated and treat appropriately and for an adequate amount of time.
- A female of childbearing age should be considered pregnant until proven otherwise with sufficient laboratory testing.
- Always screen for and treat asymptomatic bacteriuria in any pregnant patient.
- Rule out the diagnosis of testicular torsion in any patient with acute scrotal pain.
- Do not rule out ovarian torsion based on the absence of symptoms.
- Consider the diagnosis of pelvic inflammatory disease in any patient who presents with acute pelvic pain with no other explanation and treat empirically.

References

1 Pearle MS, Calhoun EA, Curhan GC. Urologic diseases in America project: urolithiasis. *J Urol* 2005; **173**(3): 848–857.
2 Chewcharat A, Curhan G. Trends in the prevalence of kidney stones in the United States from 2007 to 2016. *Urolithiasis* 2021; **49**(1): 27–39.
3 Scales CD Jr, Curtis LH, Norris RD, *et al*. Changing gender prevalence of stone disease. *J Urol* 2007; **177**(3): 979–982.
4 Alnazari M, Bakhsh A, Shaqroon HA, *et al*. Hematuria: is it useful in predicting renal or ureteral stones in patient presenting to emergency department with flank pain? *Urol Ann* 2024; **16**(1): 71–74.
5 Smith RC, Verga M, McCarthy S, *et al*. Diagnosis of acute flank pain: value of unenhanced helical CT. *AJR Am J Roentgenol* 1996; **166**(1): 97–101.
6 Lei B, Harfouch N, Scheiner J, *et al*. Can obstructive urolithiasis be safely excluded on contrast CT? A retrospective analysis of contrast-enhanced and noncontrast CT. *Am J Emerg Med* 2021; **47**: 70–73.
7 Moore CL, Carpenter CR, Heilbrun ME, *et al*. Imaging in suspected renal colic: systematic review of the literature and multispecialty consensus. *J Am Coll Radiol* 2019; **16**(9 Pt A): 1132–1143.
8 Radcliffe RS, Khan MA. Mortality associated with Fournier's gangrene remains unchanged over 25 years. *BJU Int* 2020; **125**(4): 610–616.
9 El-Qushayri AE, Khalaf KM, Dahy A, *et al*. Fournier's gangrene mortality: a 17-year systematic review and meta-analysis. *Int J Infect Dis* 2020; **92**: 218–225.
10 Koch GE, Dropkin BM, Kaufman MR. Prodromal Fournier's gangrene: a potential opportunity for early intervention. *J Urol* 2022; **208**(6): 1176–1177.
11 Dorazio J, Chiappelli AL, Shields RK, *et al*. Clindamycin Plus vancomycin versus linezolid for treatment of necrotizing soft tissue infection. Open forum. *Infect Dis* 2023; **10**(6): ofad258.
12 Stamm WE, Hooton TM. Management of urinary tract infections in adults. *N Engl J Med* 1993; **329**(18): 1328–1334.
13 Foxman B, Barlow R, D'Arcy H, *et al*. Urinary tract infection: self-reported incidence and associated costs. *Ann Epidemiol* 2000; **10**(8): 509–515.
14 Litwin MS, Saigal CS, Beerbohm EM. The burden of urologic diseases in America. *J Urol* 2005; **173**(4): 1065–1066.
15 Falagas ME, Alexiou VG, Giannopoulou KP, *et al*. Risk factors for mortality in patients with emphysematous pyelonephritis: a meta-analysis. *J Urol* 2007; **178**(3 Pt 1): 880–885. quiz 1129.
16 Gupta K, Hooton TM, Naber KG, *et al*. International clinical practice guidelines for the treatment of acute uncomplicated cystitis and pyelonephritis in women: a 2010 update by the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases. *Clin Infect Dis* 2011; **52**(5): e103–e120.
17 Fosse PE, Brinkman KM, Brink HM, *et al*. Comparing outcomes among outpatients treated for pyelonephritis with oral cephalosporins versus first-line agents. *Int J Antimicrob Agents* 2022; **59**(4): 106560.
18 Ringdahl E, Teague L. Testicular torsion. *Am Fam Physician* 2006; **74**(10): 1739–1743.
19 Anderson JB, Williamson RC. Testicular torsion in Bristol: a 25-year review. *Br J Surg* 1988; **75**(10): 988–992.
20 Melekos MD, Asbach HW, Markou SA. Etiology of acute scrotum in 100 boys with regard to age distribution. *J Urol* 1988; **139**(5): 1023–1025.
21 Collins MM, Stafford RS, O'Leary MP, *et al*. How common is prostatitis? A national survey of physician visits. *J Urol* 1998; **159**(4): 1224–1228.
22 Tracy CR, Steers WD, Costabile R. Diagnosis and management of epididymitis. *Urol Clin North Am* 2008; **35**(1): 101–108. vii.
23 Luzzi GA, O'Brien TS. Acute epididymitis. *BJU Int* 2001; **87**(8): 747–755.
24 Caesar RE, Kaplan GW. The incidence of the cremasteric reflex in normal boys. *J Urol* 1994; **152**(2 Pt 2): 779–780.
25 Nelson CP, Williams JF, Bloom DA. The cremasteric reflex: a useful but imperfect sign in testicular torsion. *J Pediatr Surg* 2003; **38**(8): 1248–1249.
26 Asgari SA, Mokhtari G, Falahatkar S, *et al*. Diagnostic accuracy of C-reactive protein and erythrocyte sedimentation rate in patients with acute scrotum. *Urol J* 2006; **3**(2): 104–108.
27 Barbosa JA, Tiseo BC, Barayan GA, *et al*. Development and initial validation of a scoring system to diagnose testicular torsion in children. *J Urol* 2013; **189**(5): 1859–1864.
28 Sheth KR, Keays M, Grimsby GM, *et al*. Diagnosing testicular torsion before urological consultation and imaging: validation of the TWIST score. *J Urol* 2016; **195**(6): 1870–1876.
29 Tracy CR, Costabile RA. The evaluation and treatment of acute epididymitis in a large university based population: are CDC guidelines being followed? *World J Urol* 2009; **27**(2): 259–263.
30 Drury NE, Dyer JP, Breitenfeldt N, *et al*. Management of acute epididymitis: are European guidelines being followed? *Eur Urol* 2004; **46**(4): 522–524; discussion 524–5.
31 Workowski KA, Bachmann LH, Chan PA, *et al*. Sexually transmitted infections treatment guidelines, 2021. *MMWR Recomm Rep* 2021; **70**(4): 1–187.
32 Ramoska EA, Sacchetti AD, Nepp M. Reliability of patient history in determining the possibility of pregnancy. *Ann Emerg Med* 1989; **18**(1): 48–50.
33 Wessel J, Buscher U. Denial of pregnancy: population based study. *BMJ* 2002; **324**(7335): 458.

34 Stengel CL, Seaberg DC, MacLeod BA. Pregnancy in the emergency department: risk factors and prevalence among all women. *Ann Emerg Med* 1994; **24**(4): 697–700.
35 Sheiner E, Mazor-Drey E, Levy A. Asymptomatic bacteriuria during pregnancy. *J Matern Fetal Neonatal Med* 2009; **22**(5): 423–427.
36 Millar LK, Cox SM. Urinary tract infections complicating pregnancy. *Infect Dis Clin North Am* 1997; **11**(1): 13–26.
37 Nicolle LE, Gupta K, Bradley SF, *et al*. Clinical practice guideline for the Management of Asymptomatic Bacteriuria: 2019 update by the Infectious Diseases Society of America. *Clin Infect Dis* 2019; **68**(10): e83–e110.
38 Anon. Urinary tract infections in pregnant individuals. *Obstet Gynecol* 2023; **142**(2): 435–445.
39 Hibbard LT. Adnexal torsion. *Am J Obstet Gynecol* 1985; **152**(4): 456–461.
40 Burnett LS. Gynecologic causes of the acute abdomen. *Surg Clin North Am* 1988; **68**(2): 385–398.
41 Bridwell RE, Koyfman A, Long B. High risk and low prevalence diseases: ovarian torsion. *Am J Emerg Med* 2022; **56**: 145–150.
42 Gupta A, Gadipudi A, Nayak D. A five-year review of ovarian torsion cases: lessons learnt. *J Obstet Gynaecol India* 2020; **70**(3): 220–224.
43 Houry D, Abbott JT. Ovarian torsion: a fifteen-year review. *Ann Emerg Med* 2001; **38**(2): 156–159.
44 Lareau SM, Beigi RH. Pelvic inflammatory disease and tubo-ovarian abscess. *Infect Dis Clin North Am* 2008; **22**(4): 693–708.

CHAPTER 3
Orthopedic Pitfalls of the Upper Extremity

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Introduction

The upper extremities are subject to a variety of traumatic and infectious insults, especially in young adult men. Estimates in the United States for an upper-extremity injury are rising, with the most common injury being fractures [1], and many of these patients will receive their initial evaluation in the urgent care setting. Although most complaints will have a straightforward presentation, the acute care clinician must be vigilant in order to avoid potential pitfalls.

Pitfall | **Failure to diagnose a posterior shoulder dislocation**

The shoulder is the most frequently dislocated joint in the body. While in 95% of cases the humeral head dislocates anterior to the glenoid (anterior dislocation) [2], it is critical to consider posterior shoulder dislocation in the differential for acute shoulder pain, even though it occurs in less than 5% of cases. Posterior shoulder dislocations are missed more than 50% of the time, both because it is uncommon and because it is difficult to diagnose on radiographs without a high index of suspicion [3]. Concomitant fractures, typically of the proximal humerus, make diagnosis even more challenging as they obscure radiographic interpretation. As many as 65% of posterior shoulder dislocations have an associated injury, including humerus fractures, reverse Hill–Sachs lesions, and rotator cuff tears [4]. If the presentation is subacute, it may be diagnosed as adhesive capsulitis or a rotator cuff tendinopathy. This can be seen in those that have microtrauma leading to posterior instability that can be insidious in onset and presentation [4]. The most salient reason for missing the diagnosis is failure to consider a posterior dislocation.

There are several soft tissue and osseous stabilizers of the shoulder that prevent posterior dislocation, but certain maneuvers can overcome these stabilizing forces, such as a direct blow, seizure, or electrocution/lightning strike [3]. The rotator cuff, glenoid labrum, and glenohumeral ligaments stabilize the humerus in the glenoid fossa [4]. The external rotators are much weaker than the internal, and thus a strong contraction of both sets, such as may happen in an electrical injury or a generalized tonic–clonic seizure, will preferentially pull the humeral head posteriorly out of its position. A posterior dislocation may also be caused by a fall onto a shoulder held in forward flexion, adducted, and in internal rotation [5]. Failure to diagnose this entity results in degenerative changes in the joint, avascular necrosis, and disability [6]. Despite the infrequent presentation, a few simple elements of the physical examination can help to suggest or rule out the diagnosis.

The affected arm will, as a rule, be held in an adducted, internally rotated position. Inspection of the shoulder in an acute posterior dislocation may show a prominent coracoid process and an anterior "void." The humeral head may be visualized or palpable posteriorly [6]. These findings may be subtle, however, and further examination of the passive and active range of motion

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of the shoulder is warranted. The patient will be unable to externally rotate the humerus, or to supinate the ipsilateral forearm, to any significant degree, either actively or passively [4].

KEY FACT | **If the patient has limited external rotation with the shoulder "locked" in internal rotation, consider posterior dislocation.**

Shoulder radiographs in the setting of acute trauma should include an anteroposterior (AP), scapular Y, and axillary lateral view. The axillary view is the most important view to evaluate the location of the humeral head in relation to the glenoid and coracoid (Figure 3.1).

Unfortunately, to obtain an ideal axillary image, the humerus must be moved into 15–20° of abduction. If the patient cannot tolerate this position, consider a Valpeau or modified trauma axial view as an alternative [7]. There are several subtle signs on the AP view, taken with the arm in neutral position, that may indicate a posterior dislocation:

- *Internal rotation of the humeral head*: The humerus may appear internally rotated, with disappearance of the greater tuberosity as it moves from the lateral to an anterior position (see Figure 3.2).
- *Light bulb sign*: This results from the fixed internal rotation of the humeral head and the symmetric appearance of the humeral head and tapering shaft (see Figure 3.2).
- *Rim sign*: The normal oval area of overlap between the humeral head and the glenoid may be widened or show diminished or uneven overlap (see Figure 3.2).

Figure 3.1 Axillary view demonstrating a posterior dislocation.

Figure 3.2 AP view showing signs of posterior shoulder dislocation.

- *Trough sign*: An impaction fracture of the humeral head causes a parallel line to be visible, just lateral to the medial border of the humeral head.
- *Vacant glenoid sign*: The anterior glenoid looks empty and may be widened by more than 6 mm (see Figure 3.2).
- *Isolated fracture of the lesser tuberosity*: This should lead one to suspect posterior dislocation of the shoulder until proven otherwise [6, 7].

If a posterior dislocation is diagnosed, transfer to the ED is warranted for evaluation, reduction under sedation, and possible orthopedic referral.

KEY FACT | **The axillary view should be ordered for all patients with potential shoulder injuries or humeral injuries as it is the most sensitive means of identifying posterior shoulder dislocation.**

Pitfall | **Failure to recognize subtle fracture findings at the elbow**

A fall directly onto the elbow or on an outstretched hand (FOOSH) is a mechanism that can lead to a fracture at the elbow. In a patient with a FOOSH injury, it is necessary to include evaluation of the elbow when examining the patient clinically and radiographically. When obtaining an x-ray of the elbow two views, including a true lateral film, should be obtained to evaluate for elevation of the anterior fat pad or the presence of a posterior fat pad. A posterior fat pad is always considered pathologic and should be assumed to represent occult fracture. It is typically indicative of an elbow joint effusion. While a small anterior fat pad is often physiologic, a large anterior fat pad is also pathologic. This is commonly known as the "sail sign" because its silhouette is similar to a boat's sail, and indicates that an effusion of the elbow joint is present [8]. If either a posterior fat pad or a large anterior fat pad is present, one should assume that an occult fracture is present and immobilize in a posterior splint with close orthopedic follow-up.

In adults, the elevation of the anterior fat pad is most indicative of radial head fracture. One meta-analysis of adults with a radiographic sail sign found that 75% had an occult fracture identified on follow-up magnetic resonance imaging (MRI). Other fracture sites include the lateral epicondyle and the olecranon [9]. Typically, a sling followed by early range of motion will be sufficient immobilization in adults with a radial head fracture [10].

In children, the elevation of the anterior fat pad is most indicative of a supracondylar fracture. A meta-analysis evaluated 10 studies with a total of 250 patients where 104 of those with a sail sign were found to have an occult fracture, 43% being supracondylar fractures in children. Other occult fractures that can be the culprit include proximal ulna, proximal radius, or lateral condyle [11]. In this case an above-the-elbow posterior slab splint with 30–40° of flexion is necessary for presumed type 1 nondisplaced supracondylar fractures in the pediatric population [12].

Pitfall | **Failure to consider septic bursitis in a patient with a swollen elbow**

Bursae are small, fluid-filled sacs that serve to facilitate motion between tendons and bone. Of the 150 bursae in the body, the olecranon is the most often affected by an inflammatory process [13]. Inflammation of these bursae can result from various infectious and noninfectious etiologies. Noninfectious bursitis can be the result of acute or chronic trauma, a crystal arthropathy, or autoimmune disease. These factors can also predispose the bursae to subsequent infection [14].

Infectious, or septic, bursitis is typically triggered by a direct percutaneous mechanism, as would happen with a traumatic injury, or by spread from an overlying cellulitis. While septic olecranon bursitis can be accompanied by a coincident septic arthritis, a joint space infection usually results from hematogenous seeding, whereas bacteremia does not usually lead to septic bursitis [15].

Most cases, 80–85%, of septic bursitis are due to *Staphylococcus aureus*, with other organisms including *Streptococcus pyogenes* and *Staphylococcus epidermidis* [16]. A chronic infection should prompt consideration of atypical infection, such as fungi [15].

> KEY FACT | **Septic bursitis, unlike septic arthritis, does not significantly affect the range of motion of the underlying joint**

Septic olecranon bursitis typically presents as a tender, swollen, and erythematous bursa overlying the elbow. The swelling is described as locally edematous, with the maximum tenderness centered on the bursa [13]. In contrast to septic arthritis, the active range of motion should not be significantly affected. Although there are elements of the history and physical examination that differentiate septic bursitis from noninfectious causes of bursitis, such as a shorter duration of symptoms, a greater degree of pain and tenderness, and increased erythema, these differences are of uncertain value for definitive diagnosis. The patient may have a fever, but this finding cannot be used to rule out septic bursitis. Perhaps more useful is the finding that the skin over a septic bursitis is consistently judged to be warmer than the contralateral noninfected elbow [16].

Labs, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and white blood cell (WBC) count, are of questionable utility as the values for septic and nonseptic bursitis overlap to a great degree, with high rates of false negatives and low predictive value [17].

The gold standard for diagnosis of a septic bursitis is a culture of the bursal fluid. However, aspiration in the acute phase comes with a risk of fistula formation. Recent literature has challenged whether this needs to occur on the initial presentation [18]. Therefore in conjunction with very close follow-up, one should consider empiric antibiotic and anti-inflammatories

management of uncomplicated septic bursitis [19]. More recent consensus statements also recommend the use of ultrasound to help distinguish cellulitis from bursitis – with bursitis having a greater volume of fluid in the bursa compared to the contralateral side, but further inquiry is required to establish guidelines based on volume [20].

If a patient has been treated empirically for septic bursitis with appropriate antibiotics and continues to have progression of swelling, erythema, and warmth, then an aspiration should be performed to identify the causative microbe and guide antimicrobial management. The procedure employs an 18 gauge needle with a 10–20 ml syringe. Use a sterile technique and clean the area with chlorhexidine or an equivalent antiseptic cleanser. The site of needle entry must be free of signs of infection, such as cellulitis. In the age of ultrasound, needle guidance with or without color Doppler can be used to aid in this procedure. The needle is directed parallel to the forearm along the lateral aspect, avoiding the ulnar nerve [20, 21]. After an anesthetic wheel is placed, the aspiration is performed with the aim of draining the bursa; "milking" of the sac may be required. A bandage and compressive dressing should be applied after the procedure. Bursal fluid should be analyzed for cell count, gram stain, culture, and, if gout or other crystalline arthropathy is suspected, crystals. It is important to keep in mind that a diagnosis cannot be made by gross visualization of the aspirate—septic fluid may be only moderately turbid, while a seemingly purulent aspirate may have a noninfectious cause, such as gout. Findings of more than 50% of polymorphonuclear leukocytes in the cell count are suggestive of septic bursa. Unlike a septic joint, there is no cutoff value for the cell count that defines septic bursitis, with values between 1,000 and 450,000 WBCs per mm^3 being cited [21].

Despite the support for diagnostic aspiration, there are concerns that complications, such as seeding of the bursa or fistulization of the tract leading to chronic drainage, may complicate this decision. There are no clear recommendations to aspirate or not aspirate, but the decision should be made after discussion with orthopedics, especially on first presentation. Our recommendation, specifically in an urgent care setting, would be to defer aspiration during the initial presentation and treat empirically with antibiotics and anti-inflammatories. Regardless of the diagnostic approach, it is important to arrange for re-evaluation within 48–72 hours [13]. If a patient appears to be acutely ill with signs of systemic infection, recommendation for blood cultures, transfer to an ED, orthopedic referral, and probable admission are advised.

KEY FACT | **All patients with a possible septic bursitis require re-evaluation within 48–72 hours**

Recommendations for empiric treatment involve 2–3 weeks of a first-generation cephalosporin. If the patient is allergic, clindamycin or trimethoprim-sulfamethoxazole can be used [21]. A number of references advise coverage for methicillin-resistant *Staphylococcus aureus* (MRSA) in patients with severe disease, past history of MRSA infection, or risk factors for MRSA colonization [20]. Outpatient treatment may be appropriate for most mild and moderate cases, but early follow-up is required in all cases to assure adequate therapy, assessment for repeated drainage, and development of systemic symptoms.

Pitfall | Failure to recognize a potential scaphoid injury

The scaphoid bone of the wrist occupies a precarious location anatomically, which explains its prominent placement among the pitfalls. The scaphoid is the most fractured carpal bone, accounting for 80% of all carpal bone fractures [22]. Diagnosis is difficult because physical exam and radiographic findings are inconsistent. However, the risks of missing this fracture are significant and include nonunion, functional morbidity, and eventual arthritic degeneration [23].

The scaphoid acts as a bridge connecting the distal radius to the wrist and is most prominent during wrist extension. These anatomic factors make the scaphoid vulnerable during a FOOSH injury, which typically occurs in wrist extension and onto the radial half of the hand [23]. Recovery of any fracture is dependent on a constant nutritional source from the blood supply; however, the blood supply varies to the different parts of the scaphoid. The lateral and distal branches of the radial artery supply the bone distally and the proximal pole relies on retrograde interosseous blood flow. This pattern predisposes fractures of the proximal pole to avascular necrosis and subsequent bony resorption [24].

All patients who sustain trauma to the distal upper extremity should receive a full musculoskeletal and neurovascular examination. This should include an evaluation for scaphoid bone injury, especially if there is

a history of a FOOSH mechanism. This evaluation comprises a few specific tests.

The best-known test is for tenderness in the anatomic snuffbox (ASB). This is the depression formed by the borders of the radial styloid, the extensor pollicis longus tendon, and the paired extensor pollicis brevis and abductor pollicis longus tendons. The scaphoid is located on the "floor" of this triangular depression (see Figure 3.3). Tenderness to palpation in the floor of the snuffbox should be treated conservatively as a fracture. This should be compared to the contralateral side, as deep palpation of this space can be uncomfortable at baseline. Another important test is axial compression of the scaphoid. The thumb is held gently in mid-abduction and extension, and force is directed axially toward the base of the thumb. Again, if this test elicits pain, the patient should be treated as having a potential scaphoid fracture. A less commonly applied, but more accurate, test is palpation for tenderness of the scaphoid tubercle [21]. This prominence of the distal scaphoid is found on the volar aspect of the wrist, along the distal wrist flexor crease, about 1 cm medial to the radial styloid. The tubercle becomes more prominent in radial deviation and when fractured the tubercle is tender [23].

Combining the tests will increase the accuracy and diagnosis based on tenderness with all three tests greatly increases specificity without sacrificing sensitivity. Nonetheless, many acute care providers elect to consider a positive finding on any of these tests as evidence for a presumptive fracture [25].

When the history and/or physical examination suggests an injury, the urgent care provider should obtain radiographs of the scaphoid.

Figure 3.3 The anatomic snuffbox (ASB). The ASB is the depression formed by the borders of the radial styloid, the extensor pollicis longus tendon, and the paired extensor pollicis brevis and abductor pollicis longus tendons.

A standard series of wrist radiographs includes AP and lateral views. If these are negative, consider adding the scaphoid view. On standard radiographs, the scaphoid will appear in an oblique position and a fracture is difficult to visualize. The scaphoid view is done with the wrist held in ulnar deviation, which rotates the scaphoid into a plane perpendicular to the x-ray beam, often revealing subtle fractures. However, about 18% of patients with a concerning history and physical examination will have a fracture despite negative initial radiographs [26].

If initial radiographs do identify a fracture, the degree of displacement should be determined, as displaced fractures will require surgical management. Consultation with a hand surgeon can expedite surgical intervention. All patients with a suspected or confirmed fracture should be placed in a short arm thumb spica splint and discharged. Studies comparing long versus short arm casts found no difference in nonunion rates [27]. All scaphoid fractures should have outpatient evaluation by a hand surgeon to evaluate the need for surgical intervention and to ensure that the fracture is healing well.

If no fracture is found but the examination suggests an occult fracture, place the patient in a short arm thumb spica splint, and arrange for reevaluation and repeat radiographs in 7–10 days. These may show sclerosis of the fracture line or other subtle, indirect signs of healing. Some authorities recommend other advanced imaging such as computed tomography (CT) or magnetic resonance imaging (MRI) within days to reduce the period of unnecessary immobilization, but if concerned when evaluating in an urgent care setting it is better to immobilize in a thumb spica splint initially [28].

KEY FACT | **Almost 20% of patients with a history and examination concerning for scaphoid fracture and negative radiographs will have a fracture at follow-up.**

Pitfall | Failure to consider clenched-fist injury during evaluation of a wound to the hand

All lacerations require meticulous examination for foreign bodies and underlying structural injuries and should be examined under the entire range of motion of the affected area. Lacerations of the hand can be the result of striking another person in the teeth, known as a clenched-fist injury (CFI) or more colloquially a "fight bite." The metacarpophalangeal (MCP) is the most

commonly involved area because of its prominence when making a fist [29].

The stereotypical patient who presents to the ED with a fight bite is a young male, typically 10–34 years of age, with an injury to his dominant hand. Often the patient will present more than a day after the injury, and may be showing frank signs of infection, as approximately 10% of fight bites get infected [30]. Frequently, patients coming to the ED with a fight bite do not initially volunteer the cause as a human bite, so it is necessary to ask directly when interviewing these patients [31].

KEY FACT | **Be sure to directly prompt the patient when interviewing them about the possibility of a "fight bite" as the etiology of their injury and consider giving a course of antibiotics, even if they deny this type of injury.**

A CFI is incurred when a flexed hand is struck against teeth and will typically involve a wound overlying either the third, fourth, or fifth MCP joint. Wounds may appear minor and CFI can occur in lacerations under 5 mm [31]. The skin overlying the MCP is thin and affords little protection of the relatively superficial extensor tendons and MCP joint space, making it more likely to be infected by oral flora (see Figure 3.4). These infections can be serious and can lead to amputation if not treated promptly and appropriately [32].

Figure 3.4 Fight bite. This patient presented 12 hours after a clenched-fist injury. Note the innocuous-appearing wound over the metacarpophalangeal joint. Courtesy of Dr. Lauren Tobias.

The evaluation should be both clinical and radiologic. After a full neurovascular and musculoskeletal exam of the hand and wrist, optimize conditions for a thorough wound evaluation. Bleeding should be controlled so that examination can be done appropriately. Small lacerations may need to be extended to fully explore underlying structures. The wound must be examined in the position in which it was injured; that is, fully flexed. A partially cut extensor tendon may be missed if the finger is examined only in extension, as the injured portion of the tendon will be located proximal to the cutaneous wound. Similarly, the MCP joint should be examined with the joint in full flexion in order to investigate for the possibility of intra-articular injury. If the joint capsule is violated, there is a higher risk for the development of septic arthritis [31].

KEY FACT | **A crucial consideration is that the wound must be examined in the position in which it was injured, which is often fully flexed, so that underlying injuries, such as a laceration of the extensor tendon, can be identified.**

Radiographs of the joint are essential and should be obtained to look for fractures of the metacarpal head or proximal phalanx, or foreign bodies such as tooth fragments. Osteomyelitis should be considered in the patient who presents with a delayed infection and x-rays should be reviewed for suggestive radiographic findings [33].

After careful examination, the wound should be copiously flushed with normal saline under pressure. Barring no injury to deeper structures, the wound should be left open to heal by secondary intention, decreasing infectious risk. The hand should be splinted in the position of comfort and tetanus immunization should be addressed. One retrospective chart review compared ED irrigation to operative management and found that irrigation in the ED is sufficient for patients presenting within 24 hours and without purulent drainage [34]. However, if there is consideration for injury to the joint capsule, tendons, or neurovascular structure, then consultation with a hand surgeon should be considered.

If there is no sign of injury beyond the skin, "prophylactic" antibiotics should be prescribed and a recheck arranged in 24–48 hours [35]. Antibiotics should also be prescribed in seemingly innocuous wounds, reducing the risk of subsequent infection. Oral antibiotics should

be active against the most common oral pathogens (e.g. streptococci, staphylococci, eikenella). Amoxicillin-clavulanate is the treatment of choice, unless there is an allergy [35].

If there are signs of injury to the tendon or bone, the patient should receive intravenous antibiotics and be evaluated by a hand surgeon, often necessitating transfer to an ED. Such patients may warrant admission, especially if there are signs of infection or functional damage, or if there are any concerns about the patient's ability to follow up. Patients with an infected CFI may be taken urgently to the operating room for further examination, debridement, and irrigation.

KEY FACT | **If a patient presents with a laceration on the dorsal side of their hand over the 3rd, 4th or 5th metacarpophalangeal joint, consider the possibility of a "fight bite." These lacerations should be thoroughly irrigated, dressed, and left open allowed to heal by secondary intention. Consider an empiric course of antibiotics.**

Conditions predisposing to long-term disability

Pitfall | Diagnosing an occult tendon injury as a "sprained" finger

Many patients present to an urgent care center after having suffered an injury to a finger. While the eventual diagnosis in many of these patients will be that of a simple "sprain," also known as a "jammed finger," with a benign prognosis, the inadequate evaluation, diagnosis, and management of certain conditions could predispose the patient to long-term disability. There are several injuries the presence of which should be investigated: central slip tear, jersey finger, mallet finger, and thumb ulnar collateral ligament injury (skier's thumb), which are described next.

Central slip tear

The extensor tendons of the finger branch into three bands: a superficial central band that inserts onto the proximal middle phalanx, and two lateral bands that insert onto the distal phalanx. Forced flexion of an actively extended proximal interphalangeal (PIP) joint may cause the central slip tendon to partially or fully tear. This tendon can also tear if an injury, such as a laceration, is overlying or near the PIP joint on the dorsal surface. Less frequently, such a tear can result from a volar dislocation of the PIP joint [36].

Initially injuries to the central slip may be difficult to diagnose. In addition to the obstacles that pain and swelling present to examination, weakness of extension of the PIP joint may be occult because the action of the lateral bands can compensate. Even with a complete central slip tendon tear, the patient may be able to fully extend the PIP joint [37]. Testing the extensor tendon can be done with the modified Elson test (Figure 3.5). Eventually, in a patient with a central slip injury the lateral bands will subluxate in a palmar direction, producing the characteristic "boutonnière" deformity. A boutonnière deformity leads to flexion of the PIP joint and hyperextension of the distal interphalangeal (DIP) joint, due to the central slip disruption in addition to the lateral band subluxation. Delay in diagnosis may preclude surgical repair and appropriate physical therapy to allow for a good functional outcome [37]. If there is clinical concern, one can splint in extension with follow-up with a hand surgeon within seven days.

KEY FACT | **Diagnosing a central slip tear requires a high degree of suspicion and detailed physical examination with specific provocative testing, as even with a complete central slip tendon tear the patient may be able to extend the proximal interphalangeal joint.**

Examination may reveal maximal tenderness at the dorsal PIP joint, as well as a loss of active range of motion and strength in extension [38]. The accuracy of the examination may be enhanced with certain techniques. The modified Elson test may be used to "isolate" the central slip [37].

If the PIP joint is held flexed at 90°, the tension of the loaded central slip prevents extensor action along the lateral bands. If the central slip is torn, however, the lateral bands can be tensioned and the DIP joint extended. Using the modified Elson test the degree of extension is easily compared between the injured and uninjured fingers [37].

If a central slip injury is suspected, the PIP joint should be splinted in extension for four to six weeks, but with the DIP and MCP joints left mobile. This position is essential to preventing palmar subluxation of the lateral extensor bands and subsequent development of a boutonnière deformity [39]. Patients should be urgently

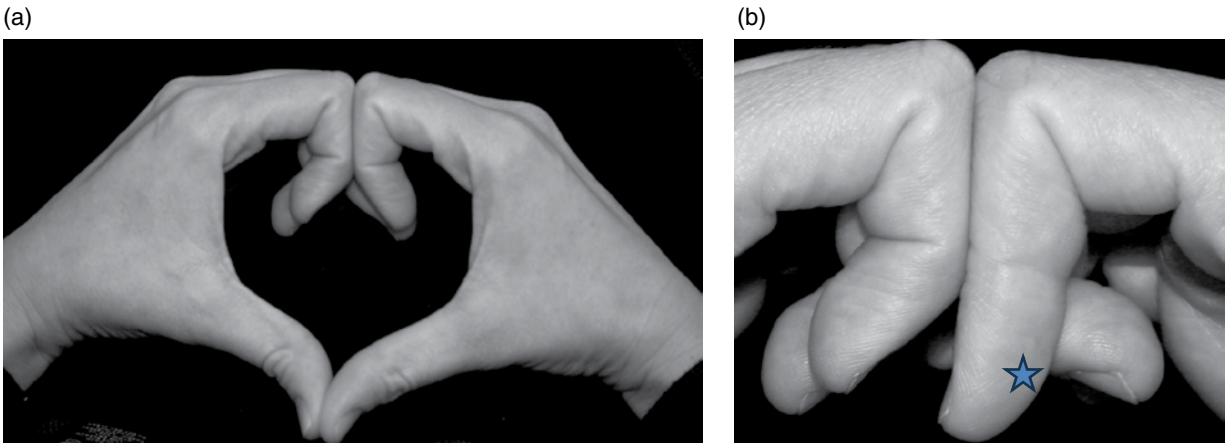

Figure 3.5 Modified Elson test. (a) The modified Elson test is performed by asking the patient to place the dorsal aspects of the middle phalanges of the injured finger and the same finger of the opposite side together with the proximal interphalangeal (PIP) joints flexed to 90°. The patient is asked to push the fingers together while trying to extend the distal interphalangeal (DIP) joints of both fingers. (b) The finger with the central slip injury will have fixed extension of the DIP joint and no active extension of the PIP joint, as seen in the left hand (starred). The injured finger is mildly extended when compared to the uninjured side.

referred to a hand surgeon if there is fracture of any size associated with the tendon injury, as they require open reduction and internal fixation [39].

KEY FACT | **Splint the proximal interphalangeal joint in full extension, with the metacarpophalangeal and distal interphalangeal joints left mobile.**

Jersey finger

Acute injuries of the ring finger that involve forced extension of a flexed DIP joint can result in injury to the flexor digitorum profundus (FDP) tendon. The colloquial name for this injury, "jersey finger," refers to a common precipitating event—an athlete grabbing another player's jersey with a flexed finger, only to have their finger forcibly pulled away, forcing the finger into hyperextension [40].

The weakest point of this tendon is just distal to the insertion on the volar base of the distal phalanx and about half of these injuries will be associated with a concomitant bony avulsion fracture [41]. The bony fragment may limit proximal retraction of the tendon. Without a bony fragment the torn tendon may retract proximally much further, and the subsequent risk of developing a contracture is much higher. Retraction of this tendon is the reason that prompt surgical referral is necessary.

On examination, there will typically be significant tenderness at the DIP joint with variable swelling [42]. The path of the FDP should be palpated to determine the point of maximum tenderness, as this could represent the distal edge of the torn tendon. AP and lateral radiographs should be obtained to look for bony avulsion.

It is necessary to specifically test the strength of the FDP, as the FDP has attachment distal to the flexor digitorum superficialis (FDS). To isolate the action of the FDP, the finger is flexed at the DIP joint while the MCP and PIP joints are held in extension by the examiner. If the patient has diminished strength on this exam, a tendon avulsion should be assumed and splinting and urgent referral arranged. If the FDP and FDS tendons are not isolated on examination, it is easy to miss a torn tendon (see Figure 3.6).

KEY FACT | **Flexion of the distal interphalangeal joint needs to be assessed in a manner that isolates the action of the flexor digitorum profundus (FDP).**

A splint should be applied before discharge. Apply a dorsal splint to the wrist and hand that maintains the wrist in 30° of flexion, the MCP in 70° of flexion, and the PIP and DIP joints in 30° of flexion [41].

Almost all injuries of the FDP will require operative management, with the best results obtained with acute

Figure 3.6 Flexor digitorum profundus (FDP) test. To isolate the flexion of the FDP, the patient is asked to flex the finger at the distal interphalangeal joint while the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints are held in extension.

intervention. Injuries without an associated avulsion fracture should have more urgent orthopedics follow-up, as surgical repair is typically preferred within seven days [43].

Mallet finger

The injury that is commonly referred to as the "mallet finger" is the result of a partial or total tear of the distal extensor tendon of the finger that attaches to the distal phalanx. This tear may involve only the tendon just proximal to the insertion on the base of the distal phalanx, or it may involve an associated bony avulsion.

The injury is classically described as a sports injury, typically when the tip of the finger is forced into hyperflexion when "jammed" against a ball. This is the most common closed tendon injury of athletes and most commonly involves the middle, ring, or small fingers [39].

The injury classically presents with a tender, swollen DIP joint as well as an extensor lag or an inability to extend the finger at the most distal portion. In addition, extensor lag may be masked through the action of the unaffected extensor central slip tendon or may become evident only later. It is necessary to specifically examine the integrity of the DIP joint extensor tendon in isolation, both actively and passively [44].

Radiographs, both AP and lateral, should be obtained to evaluate for an occult fracture. A thorough exam should also be conducted to identify a nail bed laceration, as a fracture with a nail bed laceration would be considered an open fracture (see Figure 3.7).

KEY FACT | **It is necessary to specifically examine the integrity of the distal interphalangeal joint extensor tendon in isolation.**

Closed injuries to the distal extensor tendon, with or without a small (<25% of the articular surface) avulsion fracture, should be splinted in full extension, and if available in a stack splint. Compliance is paramount to achieving the best outcome. The DIP joint needs to be splinted in extension for six to eight weeks, without interruption. In fact, when the splint is changed patients should be advised to keep their finger in extension against a surface if necessary. Any brief interruption in maintaining full extension requires another six to eight weeks in a splint. Despite this, the risk of skin breakdown requires that the splint be removed daily for cleaning and drying. Failure to comply with the splinting regimen may result in a "swan-neck" deformity, due to unopposed action of the central slip tendon, and patients should be informed of this possibility. Most mallet fingers will heal, even if treatment is delayed initially [45].

Distal extensor tendon injuries that require referral to a hand surgeon include avulsion of more than a third of the articular surface, nontrivial soft tissue damage, inability to passively extend the DIP joint, and volar subluxation of the distal phalanx [46].

Thumb ulnar collateral ligament injury, "skier's thumb"

The ulnar collateral ligament (UCL) of the thumb acts as a restraint to valgus stress applied to the thumb. It can be injured when hyperabduction and hyperextension forces are applied to the MCP joint of the thumb, such as occurs when one falls while holding onto a ski pole, hence it was termed "skier's thumb." Failure to diagnose this results in chronic instability and laxity of the thumb and eventual arthritis [47].

Typically, the patient will report tenderness at the base of the thumb and there will be bruising and swelling. An abduction stress to the MCP joint compared to the contralateral side, done at both 30° of flexion and 0°, will reveal laxity. Ideally there is a firm end point, but if the UCL is torn stress will cause distraction beyond 35° without a firm end point. A difference of 15° between the contralateral side is also concerning for a UCL tear [47].

Figure 3.7 Fracture associated with mallet finger.

An x-ray should be obtained to look for a bony avulsion of the base of the proximal phalanx of the thumb, which is seen in 20–30% of UCL tears. It occurs when the torn UCL displaces proximally and overlays the adductor aponeurosis. This is known as a Stener lesion and it typically requires surgical intervention [48]. Ultrasound is also a reliable and accurate means to diagnose UCL injury, with sensitivity of 96% [49].

UCL lesions may be managed conservatively or surgically. Urgent referral is required if there is a Stener lesion, displaced bony avulsion, gross instability, or associated plate injury. Otherwise, immobilize the joint for four to six weeks with the thumb in a position of mild flexion and slight ulnar deviation [50].

Pearls for improving patient outcomes

- Obtain axillary view radiographs if a shoulder dislocation is suspected.
- Have a high suspicion for infection in patients with warmth and swelling in olecranon bursitis, and if discharged these patients should be reevaluated promptly within 24–48 hours.
- If a scaphoid fracture is suspected, radiographs should include anteroposterior views of the wrist in ulnar deviation, also known as a "scaphoid view." If the index of suspicion is high enough to obtain this view based on history and physical exam, the patient should be discharged in a thumb spica splint.
- Examine the closed fist injury wound through the full range of motion, most crucially with the metacarpophalangeal joint in full flexion to evaluate for extensor tendon injury.
- Jersey finger most often requires surgical management, ideally within seven days. As such, isolation of the flexor digitorum profundus is necessary and the injury may present quite subtly.
- A mallet finger should be referred to a hand surgeon if an avulsion fracture exceeds a third of the articular surface, there is significant soft tissue damage, the distal interphalangeal (DIP) joint cannot be passively extended, or there is a volar subluxation of the distal phalanx.
- Advise the patient with mallet finger that if they remove the DIP joint extension splint the finger should be kept flat on a table while the splint is off, and the patient should use assistance to resplint.
- When examining for a ulnar collateral ligament (UCL) injury it is advisable to compare it to the contralateral UCL and see if there is more than 15° of difference in laxity.

References

1 Ootes D, Lambers KT, Ring DC. The epidemiology of upper extremity injuries presenting to the emergency department in the United States. *Hand (NY)* 2012; **7**(1): 18–22. https://doi.org/10.1007/s11552-011-9383-z.

2 Ladd LM, Crews M, Maertz NA. Glenohumeral joint instability: a review of anatomy, clinical presentation, and imaging. *Clin Sports Med* 2021; **40**(4): 585–599. https://doi.org/10.1016/j.csm.2021.05.001.

3 Xu W, Huang LX, Guo JJ, *et al*. Neglected posterior dislocation of the shoulder: a systematic literature review. *J Orthop Translat* 2015; **3**(2): 89–94. https://doi.org/10.1016/j.jot.2015.02.004.

4 Rouleau DM, Hebert-Davies J. Incidence of associated injury in posterior shoulder dislocation: systematic review of the literature. *J Orthop Traum* 2012; **26**(4): 246–251. https://doi.org/10.1097/BOT.0b013e3182243909.

5 Kokkalis ZT, Iliopoulos ID, Antoniou G, *et al*. Posterior shoulder fracture-dislocation: an update with treatment algorithm. *Eur J Orthop Surg Traumatol* 2017; **27**(3): 285–294. https://doi.org/10.1007/s00590-016-1840-5.

6 Perron AD, Jones RL. Posterior shoulder dislocation: avoiding a missed diagnosis. *Am J Emerg Med* 2000; **18**(2): 189–191.

7 Neep M, Aziz A. Radiography of the acutely injured shoulder. *Radiography* 2011; **17**(3): 188–192. https://doi.org/10.1016/j.radi.2011.01.006.

8 Al-Aubaidi Z, Torfing T. The role of fat pad sign in diagnosing occult elbow fractures in the pediatric patient: a prospective magnetic resonance imaging study. *J Pediatr Orthop B* 2012; **21**(6): 514–519. https://doi.org/10.1097/BPB.0b013e328355e5d8.

9 O'Dwyer H, O'Sullivan P, Fitzgerald D, *et al*. The fat pad sign following elbow trauma in adults: its usefulness and reliability in suspecting occult fracture. *J Comput Assist Tomogr* 2004; **28**(4): 562–565. https://doi.org/10.1097/00004728-200407000-00021.

10 Swensen SJ, Tyagi V, Uquillas C, *et al*. Maximizing outcomes in the treatment of radial head fractures. *J Orthop Traumatol* 2019; **20**: 15. https://doi.org/10.1186/s10195-019-0523-5.

11 Kappelhof B, Roorda BL, Poppelaars MA, *et al*. Occult fractures in children with a radiographic fat pad sign of the elbow: a meta-analysis of 10 published studies. *JBJS Rev* 2022; **10**(10). https://doi.org/10.2106/JBJS.RVW.22.00114.

12 Vaquero-Picado A, Gonzalez- MG, Moraleda L. Management of supracondylar fractures of the humerus in children. *EFFORT Open Reviews* 2018; **3**(10): 526–540. https://doi.org/10.1302/2058-5241.3.170049.

13 Blackwell JR, Hay BA, Bolt AM, *et al*. Olecranon bursitis: a systematic overview. *Should Elb* 2014; **6**(3): 182–190. https://doi.org/10.1177/1758573214532787.

14 Blumberg G, Long B, Koyfman A. Clinical mimics: an emergency medicine-focused review of cellulitis mimics. *J Emerg Med* 2017; **53**(4): 475–484.

15 Lieber SB, Fowler ML, Zhu C, *et al*. Clinical characteristics and outcomes of septic bursitis. *Infection* 2017; **45**(6): 781–786.

16 Khodaee M. Common superficial bursitis. *Am Fam Physician* 2017; **95**(4): 224–231.

17 Nchinda NN, Wolf JM. Clinical management of olecranon bursitis: a review. *J Hand Surg Am* 2021; **46**(6): 501–506. https://doi.org/10.1016/j.jhsa.2021.02.006.

18 Thomas AL, Beyde A, Sandefur BJ, *et al*. Efficacy of empiric antibiotic therapy without aspiration for septic prepatellar bursitis in emergency department patients. *Acad Emerg Med* 2022; **29**: 1027–1032. https://doi.org/10.1111/acem.14499.

19 Lieber SB, Fowler ML, Zhu C, *et al*. Clinical characteristics and outcomes of septic bursitis. *Infection* 2017; **45**: 781–786.

20 Deal JB Jr, Vaslow AS, Bickley RJ, *et al*. Empirical treatment of uncomplicated septic olecranon bursitis without aspiration. *J Hand Surg Am* 2020; **45**(1): 20–25. https://doi.org/10.1016/j.jhsa.2019.06.012.

21 Reilly D, Kamineni S. Olecranon bursitis. *J Shoulder Elb Surg* 2016; **25**(1): 158–167. https://doi.org/10.1016/j.jse.2015.08.032.

22 Fowler JR, Hughes TB. Scaphoid fractures. *Clin Sports Med* 2015; **34**(1): 37–50. https://doi.org/10.1016/j.csm.2014.09.011.

23 Carpenter CR, Pines JM, Schuur JD, *et al*. Adult scaphoid fracture. *Acad Emerg Med* 2014; **21**(2): 101–121. https://doi.org/10.1111/acem.12317.

24 Sabbagh MD, Morsy M, Moran SL. Diagnosis and management of acute scaphoid fractures. *Hand Clin* 2019; **35**(3): 259–269. https://doi.org/10.1016/j.hcl.2019.03.002.

25 Parvizi J, Wayman J, Kelly P, *et al*. Combining the clinical signs improves diagnosis of scaphoid fractures. A prospective study with follow-up. *J Hand Surg Br* 1998; **23**(3): 324–327.

26 Alshryda S, Shah A, Odak S, *et al*. Acute fractures of the scaphoid bone: systematic review and meta-analysis. *Surgeon* 2012; **10**(4): 218–229. https://doi.org/10.1016/j.surge.2012.03.004.

27 Siotos C, Asif M, Lee J, *et al*. Cast selection and non-union rates for acute scaphoid fractures treated conservatively: a systematic review and meta-analysis. *J Plast Surg Hand Surg* 2023; **57**(1–6): 16–21. https://doi.org/10.1080/2000656X.2021.2024439.

28 Sabbagh MD, Morsy M, Moran SL. Diagnosis and management of acute scaphoid fractures. *Hand Clin* 2019; **35**(3): 259–269.

29 Kennedy SA, Stoll LE, Lauder AS. Human and other mammalian bite injuries of the hand: evaluation and management. *J Am Acad Orthop Surg* 2015; **23**(1): 47–57. https://doi.org/10.5435/JAAOS-23-01-47.

30 Godoy D, Bonadeo G, Peralta H. Fight bite injuries. *Int J Emerg Med* 2002; **1**(2). https://ispub.com/IJEM/1/2/3488.

31 Rerucha CM, Ewing JT, Oppenlander KE, *et al*. Acute hand infections. *Am Fam Physician* 2019; **99**(4): 228–236.

32 Shewring DJ, Trickett RW, Subramanian KN, *et al*. The management of clenched fist "fight bite" injuries of the hand. *J Hand Surg Eur Vol* 2015; **40**(8): 819–824. https://doi.org/10.1177/1753193415576249.

33 Smith HR, Hartman H, Loveridge J, *et al*. Predicting serious complications and high cost of treatment of tooth-knuckle injuries. *Eur J Trauma Emerg Surg* 2016; **42**(6): 701–710.

34 Harper CM, Dowlatshahi AS, Rozental TD. Challenging dogma: optimal treatment of the "fight bite". *Hand (NY)* 2020; **15**(5): 647–650. https://doi.org/10.1177/1558944719831238.
35 Breed M, Fitch R. Clenched fist injury (fight bite). In: Knoop KJ, Stack LB, Storrow AB, *et al*. (eds.), *The Atlas of Emergency Medicine* (5th edn.). New York: McGraw Hill; 2021.
36 Bindra RR, Foster BJ. Management of proximal interphalangeal joint dislocations in athletes. *Hand Clin* 2009; **25**(3): 423–435.
37 Vermaak P, Devaraj V. Don't slip up! A modified technique for assessing central slip injuries. *J Hand Surg Eur Vol* 2012; **37**(9): 893–895.
38 Geoghegan L, Wormald JCR, Adami RZ, *et al*. Central slip extensor tendon injuries: a systematic review of treatments. *J Hand Surg Eur Vol* 2019; **44**(8): 825–832. https://doi.org/10.1177/1753193419845311.
39 McMurtry JT, Isaacs J. Extensor tendons injuries. *Clin Sports Med* 2015; **34**(1): 167–180. https://doi.org/10.1016/j.csm.2014.09.005.
40 Tempelaere C, Brun M, Doursounian L, *et al*. Traumatic avulsion of the flexor digitorum profundus tendon. Jersey finger, a 29 cases report. *Hand Surg Rehabil* 2017; **36**(5): 368–372.
41 Weintraub MD, Hansford BG, Stilwill SE, *et al*. Avulsion injuries of the hand and wrist. *Radiographics* 2020; **40**(1): 163–180. https://doi.org/10.1148/rg.2020190085.
42 Lapegue F, Andre A, Brun C, *et al*. Traumatic flexor tendon injuries. *Diagn Interv Imaging* 2015; **96**(12): 1279–1292. https://doi.org/10.1016/j.diii.2015.09.010.
43 Shapiro LM, Kamal RN. Evaluation and treatment of flexor tendon and pulley injuries in athletes. *Clin Sports Med* 2020; **39**(2): 279–297. https://doi.org/10.1016/j.csm.2019.12.004.
44 Lamaris GA, Matthew MK. The diagnosis and management of mallet finger injuries. *Hand (NY)* 2017; **12**(3): 223–228. https://doi.org/10.1177/1558944716642763.
45 Bachoura A, Ferikes AJ, Lubahn JD. A review of mallet finger and jersey finger injuries in the athlete. *Curr Rev Musculoskelet Med* 2017; **10**(1): 1–9. https://doi.org/10.1007/s12178-017-9395-6.
46 Cheung JP, Fung B, Ip WY. Review on mallet finger treatment. *Hand Surg* 2012; **17**(3): 439–447. https://doi.org/10.1142/S0218810412300033.
47 Madan SS, Pai DR, Kaur A, *et al*. Injury to ulnar collateral ligament of thumb. *Orthop Surg* 2014; **6**(1): 1–7. https://doi.org/10.1111/os.12084.
48 Beutel BG, Melamed E, Rettig ME. The Stener lesion and complete ulnar collateral ligament injuries of the thumb a review. *Bull Hosp Jt Dis* 2013; **77**(1): 11–20.
49 Raheman FJ, Rojoa DM, Dhingra M, *et al*. The role of ultrasonography in the assessment of ulnar collateral ligament injury of the thumb – a diagnostic test accuracy meta-analysis. *J Plast Surg Hand Surg* 2021; **55**(2): 83–95. https://doi.org/10.1080/2000656X.2020.1838295.
50 Daley D, Geary M, Gaston RG. Thumb metacarpophalangeal ulnar and radial collateral ligament injuries. *Clin Sports Med* 2020; **39**(2): 443–455. https://doi.org/10.1016/j.csm.2019.12.003.

CHAPTER 4
Orthopedic Pitfalls of the Lower Extremity

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Introduction

From 2016 to 2022 an estimated 596 injuries per 100,000 persons presented annually to emergency departments (EDs) in the United States with lower-extremity musculoskeletal injuries, with the most injured areas being the knee, ankle, and foot [1]. Many of the presenting complaints can be safely and adequately managed in an urgent care setting, but the clinician must be vigilant in evaluation of these patients in order to identify those in need of more extensive diagnostic testing, immediate referral for orthopedic care, or aggressive initial therapy.

Pitfall | **Overreliance on labs and physical exam to exclude an infectious cause for joint pain**

An atraumatic, acutely swollen, and painful joint is a common presentation to acute care centers. The etiology can range from benign to limb and life threatening. The differential for the swollen and painful joint includes degenerative arthritis, inflammatory and crystalline arthropathy, as well as septic arthritis. Septic arthritis can lead to rapid joint destruction if not quickly diagnosed and adequately treated. Mortality rate estimates range from 3 to 25% [2]. Although septic arthritis is a serious condition the initial presentation can be insidious, especially in patients who are elderly or immunocompromised (Box 4.1). Many patients lack classic findings such as fever and characteristic laboratory findings. The most common risk factor is preexisting joint space disease and/or a recent systemic infection that leads to hematogenous spread [3]. It is especially prudent to perform arthrocentesis in these patients, as one cannot rely on laboratory studies alone to rule out a septic joint.

Septic arthritis in a native joint generally occurs after hematogenous seeding of the joint during an episode of bacteremia. The bacteria enter the joint space, leading to an inflammatory reaction and associated subchondral bone destruction within a few days. The synovium is a vascular tissue that lacks a protective basement membrane, making it vulnerable to seeding from bacteremia [3]. The knee is the most commonly affected joint and is involved in 45% of cases of septic arthritis. *Staphylococcus aureus*, including methicillin-resistant *S. aureus* (MRSA), accounts for 52% of cases. Septic arthritis typically presents as a monoarticular arthritis, but can present in a polyarticular fashion up to 30% of the time [4].

It is important to remember, however, that atypical bacteria must also be considered and the differential diagnosis must remain broad. Late-stage Lyme disease must be considered as a cause of an atraumatic knee effusion in patients with potential exposures. Lyme arthritis affects the knee joint 90% of the time [5]. While research has examined potential prediction rules to help delineate between Lyme arthritis and septic arthritis, aspiration remains the standard. Lyme arthritis is characterized by a negative synovial culture but positive serologic testing for Lyme. Similarly, before establishing itself in a single joint, gonorrhea can cause an aseptic, migratory, inflammatory polyarthritis that can mimic septic arthritis. Over two-thirds of patients have fever, dermatitis, and an arthritis and diagnosis is

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Box 4.1 Risk factors associated with an increased likelihood of septic arthritis

- Prosthetic joint or recent joint surgery
- Age >80 years
- Smoking
- Skin infection, cutaneous ulceration
- History of trauma to the area over the joint
- Intravenous drug usage
- Autoimmune disease (rheumatoid arthritis, diabetes mellitus)
- Previous cartilaginous injury (osteoarthritis)
- Immunosuppression
- High-risk sexual activity

Source: Data from Earwood *et al*. [4].

often confirmed by culturing blood, synovial fluid, skin lesions, endocervix, urethra, rectum, and/or pharynx, as joint aspiration is positive less than 50% of the time.

When evaluating a patient with an acute, painful, monoarticular knee effusion, the clinician should begin with a careful history focusing on the duration of the joint pain and swelling, preceding trauma, fever, history of intravenous drug use, previous episodes, alcohol history, sexual activity, tick exposure, and immunosuppression. Physical examination should identify an effusion and, if present, the exam should be dedicated to looking for other potential causes such as fracture or meniscus or ligamentous injury. Pain with a passive short arc of motion is the most sensitive clinical exam finding for septic arthritis.

Laboratory workup can help augment clinical decision making; however, it is unable to "rule in" or "rule out" septic arthritis. Laboratory workup can include a complete blood count (CBC), an erythrocyte sedimentation rate (ESR), and a C-reactive protein (CRP). The CBC has a sensitivity of 42–90% and a positive likelihood ratio of 1.4 [2]. The clinician may also elect to obtain a serum uric acid level as hyperuricemia can predispose a patient to gout; however, serum uric acid levels can be affected by multiple factors including diet and impaired urinary excretion. While having an elevated serum uric acid level can be a predictor of gouty arthritis, up to one-third of patients with documented gout have a normal uric acid level [6]. Therefore, the presence or absence of an elevated serum uric acid level alone cannot adequately differentiate between gout and septic arthritis.

Arthrocentesis is the only reliable way to exclude septic arthritis and is the gold standard. Synovial fluid should be sent for cell count and differential, gram stain, culture, and crystal analysis. A synovial white blood cell (WBC) count of >50,000 has typically been used as the cutoff for diagnosing septic arthritis in a native joint. However, other etiologies such as gout and pseudogout can result in similar results and unfortunately, both diagnoses can occur concurrently. A synovial WBC count of >50,000 cells/mm^3 increases the likelihood of septic joint and if >100,000 cells/mm^3 the likelihood of septic joint climbs even higher [2]. However, a recent retrospective study that reviewed 40 patients with culture-positive septic arthritis found that only 13 (32.5%) had a synovial WBC count >50,000 cells/mm^3. Given this study, it is important to keep a high degree of clinical suspicion even if the cell count is not high. This is especially important to consider if a patient is immunosuppressed, as their immune response will be blunted [7]. Typically >75% of polymorphic neutrophils (PMNs) is also considered to be diagnostic of septic arthritis, but again a high degree of clinical suspicion must be maintained [2].

Arthrocentesis will also provide analysis of any crystalline material present in the joint. The two major types of synovial crystals found are negatively birefringent uric acid crystals, which indicates the patient has gout, or positively birefringent calcium pyrophosphate crystals, which indicates pseudogout. There is a concurrent rate of septic arthritis and crystalline arthropathy of about 1–2% [8]. Although this is rare, the presence of crystals does not completely exclude septic arthritis, especially in elderly patients.

Ultimately, the presence or absence of septic arthritis will be determined by the synovial fluid culture, which will not be available at the time of initial evaluation. Given the potential for morbidity from treatment delays, those patients with predictors of septic arthritis present—a history of immunosuppression, elevated ESR/CRP, elevated synovial WBC count greater than 50,000, >80% PMNs, absence of crystalline disease on arthrocentesis—should be referred to an inpatient setting for consideration of parenteral antibiotics while awaiting cultures.

In patients with a partial or complete joint replacement, consultation with their surgical provider prior to arthrocentesis is critical to ensure that their surgeon agrees with the necessity of aspiration. Arthrocentesis can seed infection into a sterile surgical

environment and thus consultation is required prior to the procedure. Typically the orthopedics team will perform these aspirations, as a positive fluid culture will have serious ramifications and often leads to revision arthroplasty. Orthopedists will also typically recommend deferring antibiotics in a stable patient until an organism can be identified.

KEY FACT | **Arthrocentesis and joint fluid analysis are required to definitively differentiate septic arthritis from other causes of a warm, erythematous, swollen and painful joint.**

Pitfall | Failure to consider gonococcal arthritis as the cause of an erythematous, swollen joint in a sexually active patient

Not all sexually transmitted infections (STIs) present with a urogenital complaint. STIs may manifest with a variety of symptoms, including joint pain, that may present to an urgent care setting. While most patients with STIs present with urogenital complaints, practitioners must be suspicious of disseminated gonococcal infection, which can lead to gonococcal arthritis. *Neisseria gonorrhoeae* is a gram-negative intracellular diplococcus that is transmitted through sexual contact. Since 2009, reported cases have been rising, with disseminated gonorrhea presenting in up to 2% of all patients infected with *N. gonorrhoeae* [9]. Disseminated gonorrhea can present with vague and nonspecific symptoms, such as arthralgias, rash, and tenosynovitis. Dissemination is more common in women than in men and classically occurs during menstruation and pregnancy. The rash is most commonly described as an erythematous maculopapular eruption, although other skin lesions including vesicles, bullae, erythema multiforme, and urticaria may be seen and the rash can be present on the palms and the soles. It is also more likely to be polyarticular [10]. Gonococcal arthritis can present with the classic triad as discussed already, with a more classic arthritis and dermatitis syndrome, but also may present without skin findings and solely with septic arthritis. In contrast to nongonococcal septic arthritis, gonococcal arthritis is more likely to involve smaller joints and to be asymmetric. It is important to keep in mind that the patient may not present with an overtly swollen joint suggestive of a suppurative arthritis and present instead with a tenosynovitis. The practitioner must maintain a high index of suspicion, as the diagnosis can again be fraught with difficulty based on the initial presentation and laboratory studies.

The patient will have a leukocytosis and elevated CRP and ESR only about 50% of the time. Arthrocentesis should be performed, but culture of *N. gonorrhoeae* requires inoculation on chocolate agar and even when done correctly, the joint fluid only reveals *N. gonorrhoeae* about 50% of the time. Furthermore, despite gonococcal arthritis being secondary to disseminated gonorrhea, less than 20% of blood cultures will be positive [11]. Because of this, it is necessary to be sure to obtain urogenital swabs or urine studies. As with septic arthritis, patients with disseminated gonorrhea and/or gonococcal arthritis should be admitted to an inpatient setting for administration of parenteral antibiotics.

KEY FACT | **Synovial cultures may be negative in patients with gonococcal arthritis.**

Pitfall | Not assessing the extensor mechanism of the knee

The integrity of the extensor mechanism is critical for normal gait and delay in diagnosis can lead to poor outcomes in patients. The extensor mechanism of the knee consists of the quadricep muscles and tendon, the patella, patellar tendon, and tibial tubercle. Injury to these structures can result in an inability to actively extend the knee. Injuries to the extensor mechanism of the knee require knee immobilization and urgent orthopedic referral, usually within seven days, for further workup and consideration of operative intervention.

Quadricep tendon ruptures (QTRs) and patellar tendon ruptures (PTRs) most commonly occur during an eccentric contraction of the quadricep muscle, usually when the foot is planted and the knee is partially flexed [12]. On the other hand, transverse patellar fractures, which disrupt the knee extensor mechanism, occur due to direct trauma onto a flexed knee. Clinical suspicion is critical as exam findings can be subtle. Initial miss rates are as high as 10–50%. If the patient is unable to complete a straight leg raise while lying supine or unable to maintain the position after passive extension, then injury to the extensor mechanism must be considered. QTRs occur 1–2 cm proximal to the superior pole of the patella and PTRs occur most commonly near the inferior pole of the patella [12]. Pain in these locations or a palpable gap may

be appreciated, but the exam can be very subtle, especially with associated effusion or swelling.

Radiographic assessment can aid in the diagnosis of patients who present with a loss of extensor mechanism. If x-rays reveal a transverse patellar fracture or tibial tubercle fracture, then the source of the extensor mechanism loss can confidently be defined, and the patient should be placed in a knee immobilizer and referred to orthopedics within five to seven days. If a fracture is not identified, then QTR or PTR must be considered. X-ray calculation of the Insall-Salvati ratio may reveal patella alta or patella baja, suggesting a tendon rupture. The Insall-Salvati ratio is calculated by measuring the patellar tendon length (LT) from the distal pole of the patella to the tibial tuberosity divided by the length of the patella (LP) from the proximal to distal pole. A normal Insall-Salvati ratio (LT/LP) is 0.8–1.2. PTR will lengthen the LT, causing the ratio to be above physiologic limits (Figure 4.1) [12].

Ultrasound can also aid in diagnosis of QTR or PTR, especially in the acute care setting. A high-frequency linear array probe directed longitudinally and transversely over the tendon will reveal hypoechoic or anechoic abnormality within the usually fibrillar-appearing tendon in the setting of a tear (Figure 4.2) [13]. Dynamic ultrasound evaluation can augment the ability to visualize tendon tears and associated intratendinous hematoma.

Clinical suspicion of QTR or PTR warrants urgent (within 7 days) orthopedic referral. Patients should be placed in a knee immobilizer, not a hinged knee brace, to prevent further tendon retraction and made non-weight bearing. For best outcomes, surgery should occur within two weeks of injury [12]. Delay in diagnosis and referral leads to poor outcomes and the need for surgical augmentation of the tendon.

KEY FACT | **Loss of the extensor mechanism of the knee may represent a quadricep or patellar tendon tear. These injuries need prompt surgical referral to optimize outcomes.**

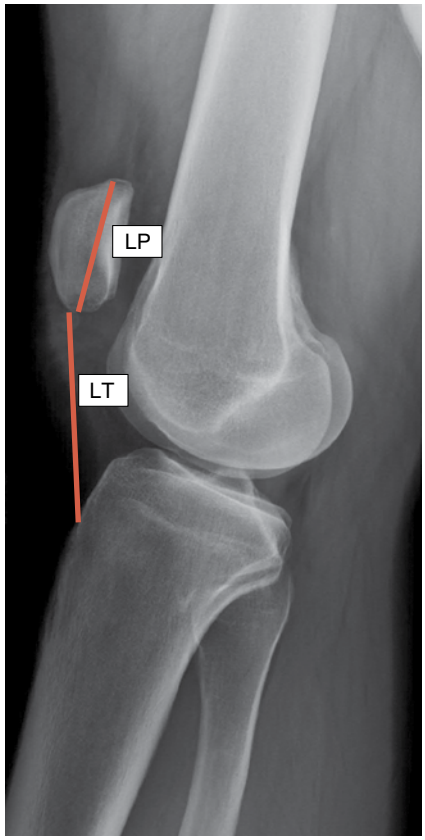

Figure 4.1 Insall-Salvati ratio suggesting a patellar tendon rupture. LT, patellar tendon length; LP, patella length.

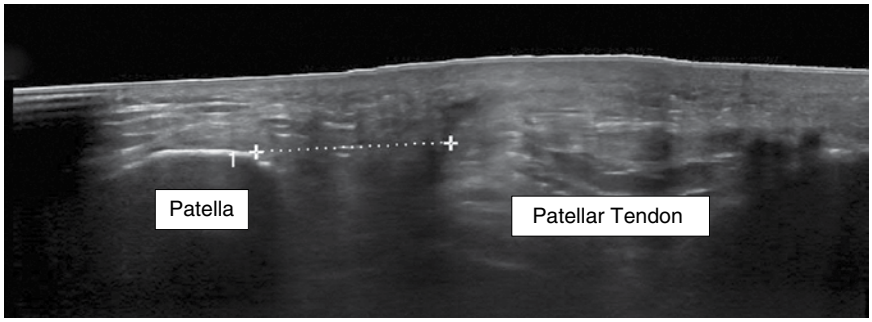

Figure 4.2 Ultrasound evaluation demonstrating patellar tendon rupture.

Pitfall | **Failing to assess the common peroneal nerve and vascular examination in ligamentous knee injury**

Knee dislocations are uncommon injuries that can cause serious morbidity if missed. It is imperative to understand that a knee dislocation can occur even in low-velocity trauma, especially if the patient is obese, and that 50% of knee dislocations are reduced before the patient arrives to an acute care center. By definition, a knee dislocation must be considered if two or more ligaments of the knee are torn [14]. If the patient's knee feels grossly unstable on examination, the clinician must seriously consider knee dislocation and vascular injury. A careful neurovascular examination must be performed, especially in this case. The major ligaments of the knee include the anterior cruciate ligament (ACL), posterior cruciate ligament, medial collateral ligament (MCL), and lateral collateral ligament. The most common pattern of ligamentous injury is of the ACL and MCL for any other combination of ligamentous injury a knee dislocation should be considered.

Knee dislocations are defined by the position of the tibia in relation to the femur. Anterior dislocations are the most common and result from rear-impact motor vehicle collisions or sporting activities [14]. Posterior dislocations occur due to dashboard injuries, fall onto a flexed knee, or varus and hyperextension of the knee [14]. Knee dislocations can cause injury to the popliteal artery due to anatomic tethering both proximally and distally. Multi-ligamentous knee injury may also result in common peroneal nerve injury. Missing either an arterial or nerve injury is catastrophic to the patient.

To assess for vascular injury, if a patient has multiligamentous instability, an ankle brachial index (ABI) should be calculated (Box 4.2). The ABI is easy to obtain, even in an urgent care setting, and is a useful tool in guiding management of known or potential vascular injuries. When emergency transport is not possible, experts agree that serial ABIs in the setting of a knee dislocation can help guide the need for transport to a facility capable of continued serial examinations and computed tomography (CT) angiography imaging [15]. An ABI <0.9 is concerning for a vascular injury and the patient requires emergency vascular surgery referral for CT angiography [16].

When clinically concerned for a knee dislocation, a neurologic exam should assess the motor and sensory function of the common peroneal nerve (Figure 4.3).

The superficial peroneal nerve supplies sensation to the lateral leg and the dorsum of the foot except for the 1st and 2nd web spaces and supplies motor innervation to the peroneal muscles that evert the ankle. The deep peroneal nerve provides sensation to the 1st and 2nd web spaces and innervates the muscles of the anterior leg that dorsiflex the ankle. Numbness in these distributions or weakness with ankle eversion or dorsiflexion is a sign of injury to the common peroneal nerve and should raise

Box 4.2 Obtaining an ankle brachial index (ABI)

$$\frac{\text{Blood pressure of the ankle}}{\text{Blood pressure of the arm}}$$

ABI ≥0.9 may be observed and have serial examinations
ABI <0.9 needs additional vascular imaging at minimum

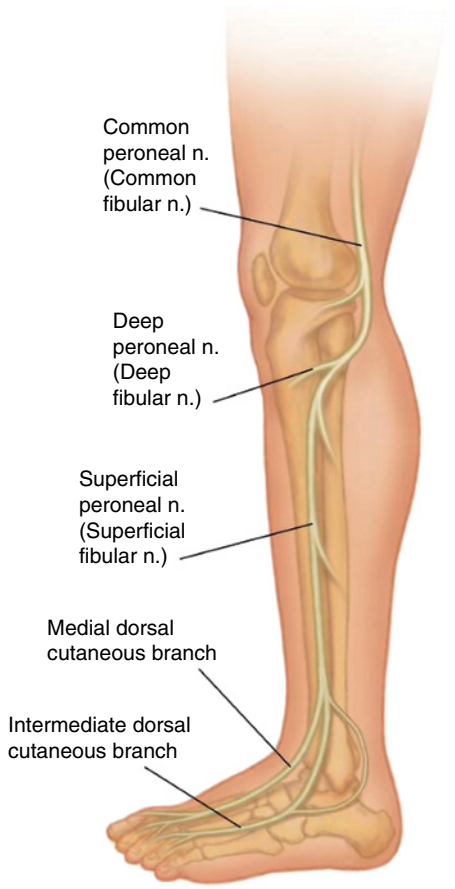
Common peroneal n. (Common fibular n.)
Deep peroneal n. (Deep fibular n.)
Superficial peroneal n. (Superficial fibular n.)
Medial dorsal cutaneous branch
Intermediate dorsal cutaneous branch

Figure 4.3 Anatomy of the nerves of the lateral lower extremity, demonstrating the location of the common peroneal nerve. *Source*: © Mayo Clinic 2013. Reproduced with permission.

concern for an associated vascular injury [16]. In the absence of vascular injury, isolated common peroneal injury in the setting of a knee dislocation requires urgent (within three to five days) orthopedic evaluation [13]. Peroneal nerve injury also typically occurs with fractures to the fibular head.

Knee dislocations without neurovascular injury should be managed in a knee immobilizer and patients should be non-weight bearing, with clear return precautions that represent evolving neurovascular injury. If the clinician has a high degree of suspicion, it is very reasonable to transfer to an ED and obtain orthopedic specialty consultation.

KEY FACT | **Knee dislocations may self-reduce prior to acute care evaluation. Multi-ligamentous knee injuries require assessment of ankle brachial index and common peroneal nerve function.**

Pitfall | **Overlooking a lipohemarthrosis**

Knee pain and effusion is a common presentation to acute care centers. The differential is broad and includes ligamentous tear, meniscal injury, osteochondral lesions, neoplasm, rheumatologic causes (including gout and pseudogout), septic arthritis, or tendon injury. Intra-articular fractures can cause a knee effusion and pain and, though they are often subtle on x-ray, failure to recognize these injuries may lead to significant long-term morbidity for the patient due to fracture displacement, chronic knee instability, and early-onset arthritis.

Intra-articular fractures includes tibial plateau, tibial eminence, and tibial tubercle fractures, and are specifically susceptible to depression and displacement, which leads to poor outcomes for patients without prompt recognition. In the acute setting, nondisplaced fracture lines on x-ray may be subtle and difficult to assess. Fortunately, a lipohemarthrosis is pathognomonic for a long bone intra-articular fracture and may be identified with ultrasound or on x-ray images [17].

In the presence of an intra-articular fracture, blood and fat from the bone marrow will leak into the joint space. Fat is less dense than blood and it will layer, creating a clear fluid level in the suprapatellar recess, which can be identified on radiographs. A cross-table lateral view is the most sensitive x-ray image for assessing for a lipohemarthrosis [18]. A double fluid–fluid level identified on x-ray is specific for a lipohemarthrosis and warrants further imaging (Figure 4.4).

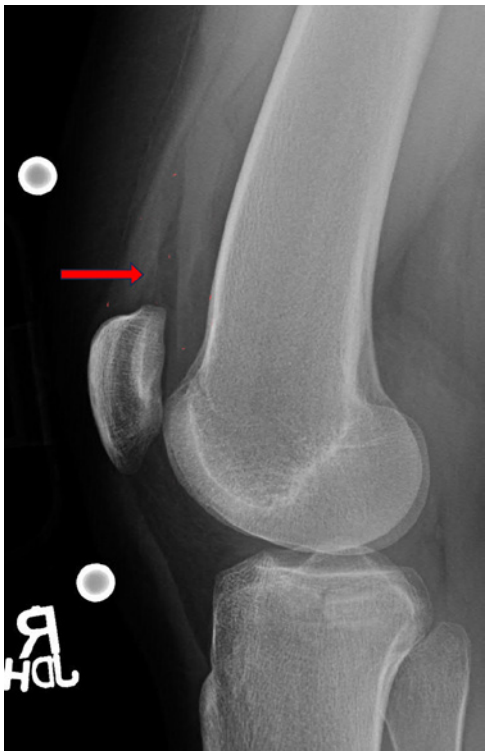

Figure 4.4 Revealing a double fluid sign (arrow) consistent with a lipohemarthrosis.

Ultrasound may also be used to assess for a lipohemarthrosis, though it is operator dependent. A linear array high-frequency probe is positioned over the quadricep tendon and suprapatellar recess. Fat will appear as a mixed echogenicity layer above the anechoic blood within the suprapatellar recess [19].

If a lipohemarthrosis is identified on ultrasound or x-ray, a CT scan should be obtained to identify the intra-articular fracture. If CT scan is not available, the patient should be made non-weight bearing, placed in a knee immobilizer, and have orthopedic follow-up within 3–5 days.

KEY FACT | **Lipohemarthrosis is suggestive of an intra-articular fracture and warrants further evaluation.**

Pitfall | **Failure to consider a syndesmotic injury in the presence of an ankle sprain**

Ankle injuries are a common complaint presenting to an acute care center. Evaluation of these patients

includes a careful history and physical examination, with a focus on the mechanism of injury and the direction of stress placed on the ankle (inversion, eversion, forced dorsiflexion, forced plantar flexion). An eversion mechanism of action should raise concern for a syndesmotic injury.

The syndesmosis of the ankle is a narrow band of connective tissue that connects the tibia and fibula, providing stability and preventing subluxation and rotation of the talus and distal fibula. Failure to recognize this injury may result in chronic instability of the ankle joint and in long-term morbidity. These injuries occur in up to 18% of ankle sprains and are commonly known as "high ankle sprains." The incidence is even higher in athletic populations, especially hockey players. The distal tibia and fibula are connected by four primary ligaments: the anterior-inferior tibiofibular ligament (AITFL), the posterior-inferior tibiofibular ligament, the interosseous ligament (IOL), and the deltoid ligament. The most common mechanism for an injury to the syndesmosis is forced internal rotation of the tibia and external rotation of the talus while the foot is planted [20]. Initially the medially located deltoid ligament is ruptured and the force is translated through the AITF ligament and IOL, and this ultimately may cause a fracture through the fibula [21]. A Maisonneuve fracture is an example of a syndesmotic injury that is characterized by a proximal fibular fracture.

Clinical exam should assess for obvious deformity followed by a good neurovascular exam. Exam findings concerning for a syndesmotic injury include pain to the AITFL and syndesmosis, pain with passive dorsiflexion and external rotation of the ankle, and pain with a lateral squeeze test. A lateral squeeze test is performed by placing one hand over the lateral fibula and one over the medial tibia just distal to the calf musculature. A compressive force will transmit through the syndesmosis and to the AITFL, suggesting a high ankle injury. If the clinician does suspect a syndesmotic injury, the entire fibula must be palpated with specific attention to the proximal fibula.

In the setting of suspected syndesmotic injury, specific attention to calculating the medial and tibiofibular clear space and tibiofibular overlap can assure that clinicians do not overlook this subtle injury, as seen in Figure 4.5.

The medial clear space is defined as the widest distance between the lateral border of the medial malleolus and medial side of the talus. On the mortise view this should be less than 4 mm, with any distance greater than 4 mm

Figure 4.5 Radiographic assessment of a syndesmotic ankle injury as seen on the mortise view of the ankle. The star shows an increased medial clear space. The black line represents a large tibiofibular clear space. The arrow reveals a lack of tibiofibular overlap.

suggestive of a deltoid ligament injury. A tibiofibular clear space (lateral clear space) greater than 6 mm on the mortise or anteroposterior (AP) view is also suggestive of a syndesmotic injury. This clear space is measured from 1 cm above the tibial plafond from the posterior tibial tubercle to the medial edge of the fibula. Tibiofibular overlap on the mortise view should be >1 mm and if <1 mm also represents injury to the interosseous membrane. If the medial clear space is >4 mm, the tibiofibular overlap <1 mm, or the tibiofibular clear space >6 mm, or a Weber B distal fibular fracture is present, then a gravity stress or manual stress view can be obtained to help further define the extent of syndesmotic injury [21, 22].

Syndesmotic injuries lead to ankle instability and require splinting and urgent orthopedic follow-up. Patients with suspected syndesmotic injuries should be immobilized in a stirrup splint and made non-weight bearing until surgical consultation can be obtained.

KEY FACT | **Syndesmotic ankle injuries are unstable ankle injuries that require close follow-up with orthopedics.**

Pitfall | **Failure to consider Achilles tendon rupture in an ambulatory patient**

While evaluating a patient with an acutely injured ankle or lower extremity it is essential to evaluate the Achilles tendon for potential injury. The Achilles tendon is the large tendon connecting the gastrocnemius and soleus muscles to the calcaneus. It is the strongest and thickest tendon in the human body and functions as the primary plantar flexor of the ankle. Achilles tendon rupture predominantly affects middle-aged men participating in athletic activities, so-called weekend warriors. Rupture occurs during acute deceleration or acceleration when the tendon is placed on an eccentric load. While its presence may be obvious to the acute care provider in certain cases, 20–25% of patients may present similarly to a simple sprained ankle and the rupture will be missed on initial evaluation [23]. Patients may be unable to ambulate following the injury and are often unable to walk on their toes and plantarflex against resistance; however, the patient may be able to do this if the other plantar flexors are intact [24]. This, in conjunction with the fact that the patient's pain may have lessened or completely resolved, results in missed or delayed diagnosis of this injury.

There are several key historical points and examination findings to suggest an Achilles tendon rupture. Clarifying the mechanism suggests the injury, as does a history of hearing a "popping sound" and the patient feeling as if they have been kicked in the leg. Some patients have a deformity over the posterior calf when comparing one extremity to the other. However, hematoma formation following acute rupture may make the extremities appear roughly symmetric [23].

Diagnosis of this injury requires that the patient be examined in a prone position with the ankles extending off the end of the examination table. Assessment of plantar flexion can be misleading, as an intact tibialis posterior may allow for function that masks the injury. The Thompson test is the most useful exam in trying to identify a rupture of the Achilles tendon. While the patient is a prone position and the knee flexed to 90°, the examiner should squeeze the calf gently and observe the affected extremity for the presence of plantar flexion in response to the squeeze. The presence of partial plantar flexion implies partial integrity of the tendon, while a lack of plantar flexion suggests complete tendon rupture; this is considered a positive Thompson test. This test has been shown to have a sensitivity of 96% and a specificity of 93% in patients with surgically proven Achilles tendon tears [25, 26]. However, this is only in acute ruptures and does not apply to cases of chronic rupture [27].

In cases where the diagnosis remains uncertain following careful physical examination, radiographic studies including ultrasound may be performed while in the urgent care setting to reveal a hypoechoic incongruency within the Achilles tendon, representing a tear. Dynamic evaluation under ultrasound can further improve the sensitivity of diagnosis. Magnetic resonance imaging (MRI) is definitive for the diagnosis; it is usually obtained in follow-up, but should ideally be obtained within seven days.

Patients with suspected Achilles tendon tear should be splinted in plantar flexion in a posterior splint or be placed in a controlled ankle movement (CAM) walking boot with an adjustable heel wedge, with instructions to be totally non-weight bearing. Close orthopedic follow-up within one week should be arranged to help guide conservative versus surgical management.

KEY FACT | **Patients with an Achilles tendon rupture may still be able to ambulate and actively plantar flex the affected lower extremity.**

Pitfall | **Failure to identify a Lisfranc fracture-dislocation on plain radiographs of the foot**

A Lisfranc fracture-dislocation can occur in the setting of high-energy trauma or via a low-energy mechanism in which the foot sustains an axial load and rotation is applied to the midfoot with the foot in plantar flexion. This diagnosis must be considered in patients with a swollen, painful midfoot, especially if the patient is unable to bear weight or has ecchymosis to the plantar midfoot [28]. Up to 20% of Lisfranc injuries are missed and diagnosed as a simple foot sprain, which can lead to serious morbidity including post-traumatic osteoarthritis or midfoot deformities [29].

The tarsometatarsal joint represents the junction of the forefoot and midfoot, with the osseous structures consisting of the bases of the five metatarsals, the three cuneiform bones, and the cuboid. Transverse ligaments connect the bases of the second through fifth metatarsals, while the Lisfranc ligament connects the medial cuneiform to the base of the second metatarsal. This ligament courses from the plantar aspect of the medial

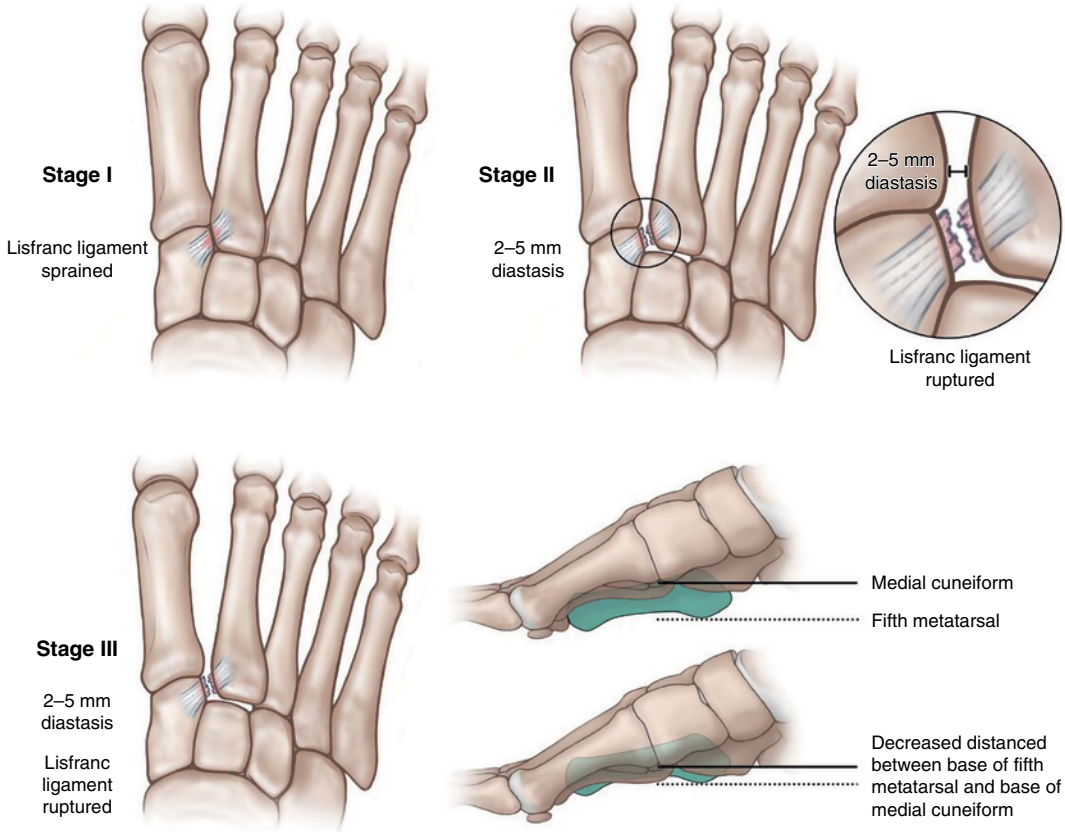

Figure 4.6 Anatomy of the Lisfranc ligament and description of injury. *Source*: © Mayo Clinic.

cuneiform to the base of the second metatarsal. The lack of an intermetatarsal ligament between the first and second metatarsals may predispose the Lisfranc ligament to injury [28]. Figure 4.6 illustrates the anatomic relationships of the bony structures forming the tarsometatarsal joint. The joint as a whole helps to maintain stability of the midfoot.

One commonly described mechanism of a Lisfranc injury is a direct blow to the foot or a significant axial load, which might occur in crush injuries, a motorcycle accident, a fall from a height, or sporting activities. An indirect force, such as a longitudinal force applied to a plantarflexed foot, can also result in a Lisfranc injury. Physical examination findings may be variable, however there is generally a marked amount of swelling and pain in the midfoot. Some patients will be completely unable to bear weight whereas others will have a relatively preserved ability to ambulate, despite significant pain. The acute care provider should palpate for tenderness along the location of the ligament between the first and second tarsometatarsal joints, which, if present, suggests underlying injury. Provocative exam testing includes the piano key test, the Lisfranc squeeze test, and the midfoot pronation and abduction test:

- *Piano key test*: The midfoot and hindfoot are manually secured and a plantar force is applied to the individual metatarsal head (as if one were striking a piano key) [30].
- *Lisfranc squeeze test*: This is performed when the examiner places the palm on the dorsum of the midfoot and applies a compressive force with the thumb and digits [31].
- *Midfoot pronation and abduction test*: This is undertaken by stabilizing the hindfoot and simultaneously pronating and abducting the forefoot to elicit pain [31].

The plantar surface of the foot should be examined carefully, as the presence of plantar ecchymosis is a pathognomonic finding and should raise the clinician's suspicion of a tarsometatarsal joint injury [28].

Plain radiographs are always part of the evaluation of an injured foot and include the AP, lateral, and oblique views [32]. If there is suspicion of a Lisfranc injury

following physical examination and history, it is important to obtain bilateral weight-bearing views. This allows for comparison between the two and weight-bearing will cause displacement to reveal the subtle ligament injury.

KEY FACT | **Up to 20% of patients with a Lisfranc injury will have negative initial radiographs.**

Radiographic signs of a Lisfranc injury include the following (Figure 4.7):

- The medial border of the second metatarsal not aligning with the medial border of the middle cuneiform.
- More than 2 mm of displacement between the medial cuneiform and the base of the second metatarsal.
- Loss of alignment between the medial edge of the cuboid bone and fourth metatarsal on the oblique view.
- Fleck sign, which is an avulsion fragment from the lateral edge of the first medial cuneiform.

If a Lisfranc injury is identified, the patient should be made totally non-weight bearing, placed in a CAM boot, and have orthopedic follow-up within one week. In the case of negative radiographs but with high clinical suspicion, the patient should similarly be immobilized and made non-weight bearing until further imaging can be obtained. Typically an MRI is obtained in the outpatient setting to visualize the ligamentous complex.

KEY FACT | **Failure to recognize and treat Lisfranc injuries can lead to devastating long-term outcomes for patients.**

Pitfall | Failing to differentiate between avulsion fractures of the fifth metatarsal and Jones fractures of the fifth metatarsal

Metatarsal fractures account for 35% of foot fractures, with 80% being minimally displaced or nondisplaced. Most commonly the fifth metatarsal is affected [33]. If the provider can elicit tenderness over the fifth

Figure 4.7 A left foot Lisfranc injury, defined by the medial border of the second metatarsal not being aligned with the medial border of the middle cuneiform and more than 2 mm of displacement between the medial cuneiform and the base of the second metatarsal. The arrow indicates widening at the Lisfranc joint.

metatarsal, radiographic views of the foot should be obtained. The cortex of the fifth metatarsal is thinner than that of other metatarsals and it has multiple ligament and capsular attachments so that significant forces can fracture the bone. Typically these fractures are divided into three zones. A zone one (most proximal) fracture results in an avulsion-type fracture that occurs secondary to forefoot supination with plantar flexion. That mechanism pulls the lateral band of the plantar fascia and the peroneus brevis, resulting in a transverse to slightly oblique fracture. A Jones fracture occurs at the junction of the diaphysis and metaphysis, at zone two, and results typically from a large adduction force applied to the forefoot with the ankle plantar flexed (Figure 4.8). Lastly, the third zone is the site of diaphyseal stress fractures, typically secondary to an overuse mechanism, but is less likely to present to an acute care setting [33].

A proximal tubercle avulsion fracture that is nondisplaced can typically be placed in a CAM boot or hard-soled shoe with weight-bearing status as tolerated and follow-up with primary care or orthopedic surgery within two weeks. If the fracture fragment is displaced more than 3 mm or if the fragment is rotated, the patient should be placed in a CAM boot with a non-weight-bearing status and orthopedic follow-up within one week [34].

Jones fractures are high-risk fracturs that occur in the "watershed" area for blood supply, which makes the fracture susceptible to nonunion. To minimize potential complications, these patients need to be strictly non-weight bearing in a CAM boot, followed by close orthopedic consultation within one week. If the patient cannot adhere to strict non-weight bearing, they should be placed in a posterior splint [34].

Stress fractures should be placed in a CAM boot, and patients should be made non-weight bearing and referred to orthopedic surgery.

KEY FACT | **Jones fracture of the fifth metatarsal is prone to nonunion and malunion and it is essential that these patients be properly immobilized and be made non-weight bearing.**

Pearls for improving patient outcomes

- Patients with an erythematous, swollen, painful joint plus a joint effusion should undergo arthrocentesis to rule out septic arthritis.

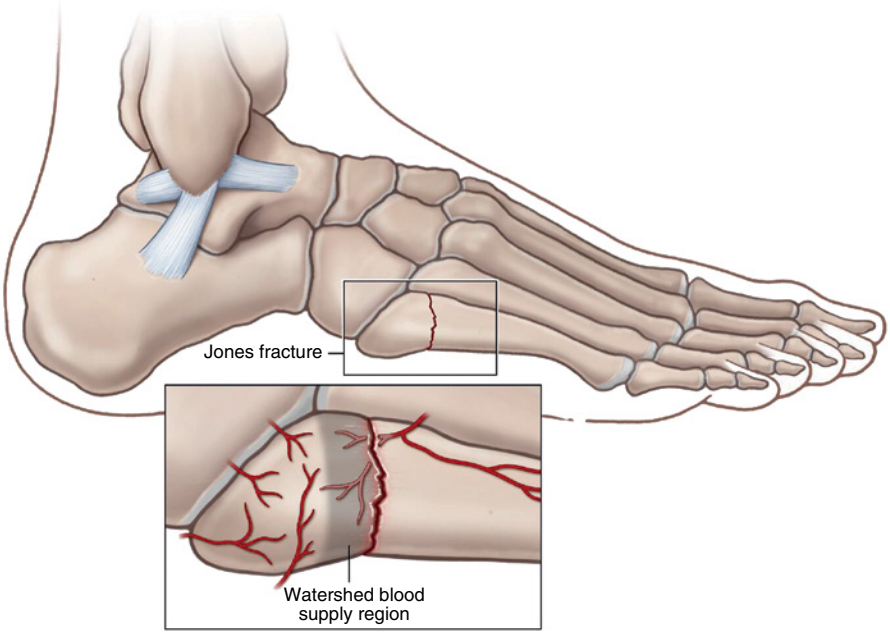

Figure 4.8 The watershed vascular supply at the base of the fifth metatarsal. *Source*: © Mayo Clinic.

• Patients with a loss of extensor mechanism of the knee, such as a quadricep tendon or patellar tendon injury, should be placed in a knee immobilizer with the knee in full extension. This requires prompt orthopedic follow-up as it may need to be repaired surgically.
• A lipohemarthrosis represents an intra-articular fracture.
• In patients with an ankle injury, palpate the proximal fibula to avoid missing a syndesmotic injury or Maisonneuve fracture, which requires different treatment and disposition than an ankle sprain or isolated fracture.
• Loss of the anatomic relationships between the medial aspect of the second metatarsal and middle cuneiform, as well as loss of the relationship between the medial aspect of the fourth metatarsal and cuboid on plain radiographs, should raise the suspicion of a Lisfranc injury.

References

1 Albright JA, Testa EJ, Testa JW, *et al*. Trends in lower extremity injuries presenting to emergency departments during the COVID-19 pandemic. *J Emerg Med* 2022; **63**(2): 169–177. https://doi.org/10.1016/j.jemermed.2022.04.032.

2 Long B, Koyfman A, Gottlieb M. Evaluation and management of septic arthritis and its mimics in the emergency department. *West J Emerg Med* 2019; **20**(2): 331–341. https://doi.org/10.5811/westjem.2018.10.40974.

3 Ross JJ. Septic arthritis of native joints. *Infect Dis Clin N Am* 2017; **31**(2): 203–218. https://doi.org/10.1016/j.idc.2017.01.001.

4 Earwood JS, Walker TR, Sue GJC. Septic arthritis: diagnosis and treatment. *Am Fam Physician* 2021; **104**(6): 589–597.

5 Orczyk K, Świdrowska-Jaros J, Smolewska E. When a patient suspected with juvenile idiopathic arthritis turns out to be diagnosed with an infectious disease – a review of Lyme arthritis in children. *Pediatr Rheumatol Online J* 2017; **15**(1): 35.

6 Dalbeth N, Gosling AL, Gaffo A, *et al*. Gout. *Lancet* 2021; **397**(10287): 1843–1855. https://doi.org/10.1016/S0140-6736(21)00569-9. Erratum in *Lancet* 2021;**397** (10287):1808.

7 Rasmussen L, Bell J, Kumar A, *et al*. A retrospective review of native septic arthritis in patients: can we diagnose based on laboratory values? *Cureus* 2020; **12**(6): e8577. https://doi.org/10.7759/cureus.8577.

8 Colaco C, Dotel R. Coexisting polyarticular septic arthritis, gout and pseudogout. *BMJ Case Rep* 2018; **2018**: bcr2018226810. https://doi.org/10.1136/bcr-2018-226810.

9 Estrada J, Sergent S, Ashurst J. Disseminated gonorrhea. *Clin Pract Cases Emerg Med* 2019; **4**(1): 83–84. https://doi.org/10.5811/cpcem.2019.9.44272.

10 Genes N, Chisolm-Straker M. Monoarticular arthritis update: current evidence for diagnosis and treatment in the emergency department. *Emerg Med Pract* 2012; **14**(5): 1–19; quiz 19–20.

11 Ohl C, Forster D. Infectious arthritis of native joints. In: Mandell GL, Bennett JE, Churchill DR (eds.), *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases* (8th edn., vol. **1**). Philadelphia (PA): Saunders, Elsevier; 2015: pp. 1302–1317.

12 Ibounig T, Simons TA. Etiology, diagnosis and treatment of tendinous knee extensor mechanism injuries. *Scand J Surg* 2016; **105**(2): 67–72. https://doi.org/10.1177/1457496915598761.

13 Ogle K, Mandoorah S, Fellin M, *et al*. Point-of-care ultrasound diagnosis of bilateral patellar tendon rupture. *Clin Pract Cases Emerg Med* 2020; **4**(1): 29–31. https://doi.org/10.5811/cpcem.2019.10.44194.

14 Gimber LH, Scalcione LR, Rowan A, *et al*. Multiligamentous injuries and knee dislocations. *Skeletal Radiol* 2015; **44**(11): 1559–1572. https://doi.org/10.1007/s00256-015-2169-6.

15 Mills WJ, Barei DP, McNair P. The value of the ankle-brachial index for diagnosing arterial injury after knee dislocation: a prospective study. *J Trauma* 2004; **56**(6): 1261–1265. https://doi.org/10.1097/01.ta.0000068995.63201.0b.

16 Dy CJ, Inclan PM, Matava MJ, *et al*. Current concepts review: common peroneal nerve palsy after knee dislocations. *Foot Ankle Int* 2021; **42**(5): 658–668. https://doi.org/10.1177/1071100721995421.

17 Bonnefoy O, Diris B, Moinard M, *et al*. Acute knee trauma: role of ultrasound. *Eur Radiol* 2006; **16**(11): 2542–2548. https://doi.org/10.1007/s00330-006-0319-x.

18 Davis DL, Vachhani P. Traumatic extra-capsular and intra-capsular floating fat: fat-fluid levels of the knee revisited. *J Clin Imaging Sci* 2015; (5): 60. https://doi.org/10.4103/2156-7514.170729.

19 Rippey J. Ultrasound for knee effusion: lipohaemarthrosis and tibial plateau fracture. *Australas J Ultrasound Med* 2014; **17**(4): 159, 166. https://doi.org/10.1002/j.2205-0140.2014.tb00239.x.

20 Porter DA, Jaggers RR, Barnes AF, *et al*. Optimal management of ankle syndesmosis injuries. *Open Access J Sports Med* 2014; **5**: 173–182. https://doi.org/10.2147/OAJSM.S41564.

21 He JQ, Ma XL, Xin JY, *et al*. Pathoanatomy and injury mechanism of typical Maisonneuve fracture. *Orthop Surg* 2020; **12**(6): 1644–1651. https://doi.org/10.1111/os.12733.

22 Gomes YE, Chau M, Banwell HA, *et al*. Diagnostic accuracy of the Ottawa ankle rule to exclude fractures in acute ankle injuries in adults: a systematic review and meta-analysis. *BMC Musculoskelet Disord* 2022; **23**(1): 885. https://doi.org/10.1186/s12891-022-05831-7.

23 Holm C, Kjaer M, Eliasson P. Achilles tendon rupture—treatment and complications: a systematic review. *Scand J Med Sci Sports* 2015; **25**(1): e1–e10.

24 Singh D. Acute Achilles tendon rupture. *BMJ* 2015; **351**: h4722. https://doi.org/10.1136/bmj.h4722.
25 Maffulli N, Via AG, Oliva F. Chronic Achilles tendon rupture. *Open Orthop J* 2017; **11**: 660–669. https://doi.org/10.2174/1874325001711010660.
26 Schwieterman B, Haas D, Columber K, *et al*. Diagnostic accuracy of physical examination tests of the ankle/foot complex: a systematic review. *Int J Sports Phys Ther* 2013; **8**(4): 416–426.
27 Shamrock AG, Dreyer MA, Varacallo M. Achilles tendon rupture. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing; 2023. https://www.ncbi.nlm.nih.gov/books/NBK430844/. Accessed July 18, 2024.
28 Clare MP. Lisfranc injuries. *Curr Rev Musculoskelet Med* 2017; **10**(1): 81–85. https://doi.org/10.1007/s12178-017-9387-6.
29 Lau S, Bozin M, Thillainadesan T. Lisfranc fracture dislocation: a review of a commonly missed injury of the midfoot. *Emerg Med J* 2017; **34**: 52–56.
30 Keiserman LS, Cassandra J, Amis JA. The piano key test: a clinical sign for the identification of subtle tarsometatarsal pathology. *Foot Ankle Int* 2003; **24**: 437–438.
31 Beutler A, Taylor C. (2023). Tarsometatarsal (Lisfranc) joint complex injuries. https://medilib.ir/uptodate/show/13795. Accessed July 18, 2024.
32 Mascio A, Greco T, Maccauro G, *et al*. Lisfranc complex injuries management and treatment: current knowledge. *Int J Physiol Pathophysiol Pharmacol* 2022; **14**(3): 161–170.
33 Bowes J, Buckley R. Fifth metatarsal fractures and current treatment. *World J Orthop* 2016; **7**(12): 793–800. https://doi.org/10.5312/wjo.v7.i12.793.
34 Bica D, Sprouse RA, Armen J. Diagnosis and management of common foot fractures. *Am Fam Physician* 2016; **93**(3): 183–191.

CHAPTER 5
Orthopedic Pitfalls: Pediatrics

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Introduction

Pediatric orthopedic injuries are challenging in both diagnosis and disposition. Young children are difficult to examine due to their anxiety and inability to articulate their location of pain or the mechanism of injury. Maintaining vigilance for the potential for child abuse in the setting of pediatric musculoskeletal trauma complicates assessment. Pediatric joints differ from those of an adult in that a child's ligaments are stronger than the bone. This leaves the relatively weak cartilaginous growth plate or apophysis as an area of least resistance, resulting in a fracture, such as an avulsion fracture, rather than a sprain. With multiple centers of developing ossification in a child and differing ages of physeal closure, assessing injuries on pediatric radiographs can be challenging. Given the difficulty with the history, physical examination, and radiologic evaluation of children, one must be diligent to avoid the pitfalls in pediatric orthopedics.

Pitfall | **Missing a growth plate fracture**

Pediatric fractures frequently involve shearing or torsional forces, which result in fractures through the cartilaginous growth plate (physis) in nearly a third of pediatric fractures [1]. Most commonly this occurs in the distal tibia or distal radius [2]. The Salter–Harris (SH) classification system is used to describe growth plate fractures (Table 5.1) [3]. This is important because the likelihood of growth plate arrest increases with progression of the SH type, from 3% for SH type 1 fractures to 13% for SH type 4 fractures. SH type 5 fractures are the result of severe impaction injury that typically is diagnosed in hindsight when growth arrest is detected [4]. SH type 1 fractures are subtle on radiographs and present with tenderness and potentially a slight widening of the growth plate, which may not be initially apparent (Figure 5.1) [5]. Therefore, any patient with tenderness at the growth plate following trauma should be treated for a possible fracture regardless of the x-ray findings [5]. The child should be splinted and have repeat imaging performed at two weeks to evaluate for any possible callus formation to indicate a fracture.

KEY FACT | **A child with point tenderness at their growth plate should be splinted and treated as if fractured, regardless of x-ray findings.**

Pitfall | **Missing a buckle fracture in a child**

A buckle, or torus, fracture of the distal forearm is the most common of all childhood extremity fractures [6]. This type of fracture typically results from a fall on an outstretched hand (FOOSH) mechanism. The force of impact causes a compression of the junction between the porous metaphysis and the denser diaphysis of the child's forearm, causing buckling of the cortex [6]. By definition, these fractures should occur more than 1 cm proximal to the distal radius physis and have less than

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Table 5.1 Salter–Harris classification of pediatric fractures.

Type	Description	Mnemonic SALTR
1	Fracture through the growth plate	**S**ame (no change in x-ray)
2	Fracture through the growth plate and away from the joint space (toward the metaphysis)	**A**way (from the joint)
3	Fracture through the growth plate and toward the joint space (toward the epiphysis)	**L**ower (into the joint)
4	Fracture through both epiphysis and metaphysis, transecting the growth plate	**T**hrough both (above and lower)
5	Complete impaction of the growth plate, usually not detected until growth arrest becomes apparent	**R**est (of the bone)

Source: Data from Salter and Harris [3].

10° of angulation [6]. Those fractures more distal and more angulated may represent a physeal fracture and are higher risk for complications, including growth arrest. Distal radius buckle fractures may present with only point tenderness at the fracture site. One must keep a high clinical suspicion due to lack of other signs and symptoms, such as swelling and arm disuse, that typically are seen with fractures. Given these minimal symptoms at the time of evaluation, it is estimated that over 20% of these fractures are initially missed, though there is rarely any complication. Radiographs of the forearm should include two views to fully assess for possible injury. Buckle fractures occur at the flare of the metaphysis. This area should be continuous and smooth. One way to better explain this is to visualize a marble rolling up the metaphysis; if it encounters any bumps on the way, that would be indicative of a fracture. It is important to closely examine the lateral film for similar smoothness, since some fractures will involve solely the dorsal cortex, making it visible only on the lateral view (Figure 5.2).

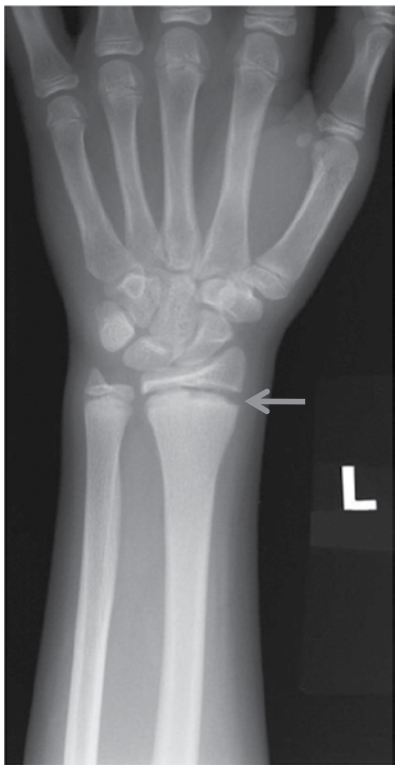

Figure 5.1 Salter–Harris type 1 fracture of the distal radius in a nine-year-old female. Note the slight widening of the growth plate of the radius (arrow).

Buckle fractures are inherently stable and are not at risk for later displacement. Once the fracture is detected, immobilization can range from rigid casting to removable wrist splints. Recent literature has shown that removable wrist splints result in quicker recovery and improved range of motion and strength without worsening displacement or angulation [7]. Patients should be immobilized for two to four weeks and refrain from sporting activities for at least six weeks [7].

KEY FACT | **Over 20% of buckle fractures are missed on initial visit.**

Pitfall | **Failure to recognize and reduce a nursemaid's elbow**

Nursemaid's elbow is a common pediatric injury that occurs when there is a sudden pull on the child's outstretched, pronated arm. This typically occurs in

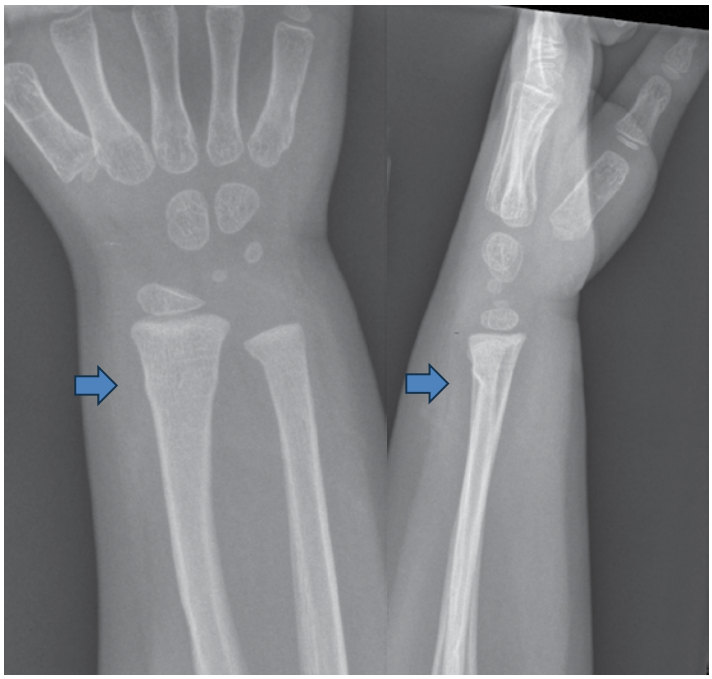

Figure 5.2 Anteroposterior (AP) and lateral forearm of a radial buckle fracture (arrows). The AP view reveals a cortical buckling. The lateral view has an abrupt change in angle of the dorsal cortex of the metaphysis, indicative of a buckle fracture.

children younger than six years. The pulling force dislocates the annular ligament that usually circles the head of the radius superiorly and dislocation traps it between the radius and the capitellum of the humerus [8]. To minimize pain, the child will naturally hold the arm close to the body, flexing at the elbow and keeping the forearm pronated. The child is usually apprehensive, but there is no true pain, swelling, or bruising about the elbow or forearm, making diagnosis difficult. Radiographs of the elbow are only necessary if there is a concerning history for fracture or any swelling, deformity, or bruising. When radiographs are obtained, they are frequently normal. Recent literature supports the use of ultrasound to confirm the diagnosis [8]. To actively reduce the dislocation, two methods are suggested: hyperpronation (Figure 5.3) and supination/flexion. The hyperpronation method has been found to be less painful in children and more successful on the first attempt [9]. With the reduction maneuver a palpable click is felt over the radial head. The child should start using the arm within 5–10 minutes after a successful reduction. Parental instruction on avoidance of picking the child up or swinging them by the arms is important to decrease recurrence. Despite these instructions, nursemaid's elbow recurs in about 25% of children [10].

KEY FACT | **Nursemaid's elbow is a clinical diagnosis; x-rays are not necessary.**

Pitfall | **Failure to identify subtle findings of a supracondylar fracture**

The interpretation of pediatric elbow radiographs can be challenging, as the normal centers of ossification can be mistaken for fractures. The centers of ossification of the pediatric elbow appear in a reliable pattern according to age (Figure 5.4), although there is variability as to when these appear.

A subtle clue to diagnosing a supracondylar humerus fracture of the elbow is the presence of a posterior fat pad. A posterior fat pad seen at the distal humerus is indicative of a joint effusion or hemarthrosis (Figure 5.5). In the setting of trauma, a posterior fat pad is always pathologic and is pathognomonic for a type 1 supracondylar fracture (nondisplaced or minimally displaced humerus fracture).

Figure 5.3 Hyperpronation method of nursemaid's elbow reduction. With the child in the parent's lap, sit facing the child. Gently hold the child's elbow, placing your thumb on the child's radial head, then, with your other hand, grasp the child's pronated wrist. With moderate force and speed, hyperpronate the child's forearm, turning the child's thumb down and around until resistance is felt. A palpable pop should be felt about the elbow when reduction occurs.

Figure 5.5 Lateral elbow radiograph at four years of age. Note the posterior fat pad sign (arrow), indicative of an occult supracondylar fracture.

Another way to detect subtle misalignment of the elbow is to assess the capitellum for displacement by using the anterior humeral line. A line drawn down the anterior cortex of the humerus on the lateral x-ray should transect the middle third of the capitellum. Usually, if there is displacement of the capitellum, this line will intersect more anteriorly on the capitellum. The neck of the radius should point directly toward and transect the center of the capitellum in all views. In uncertain cases a comparison view should be obtained.

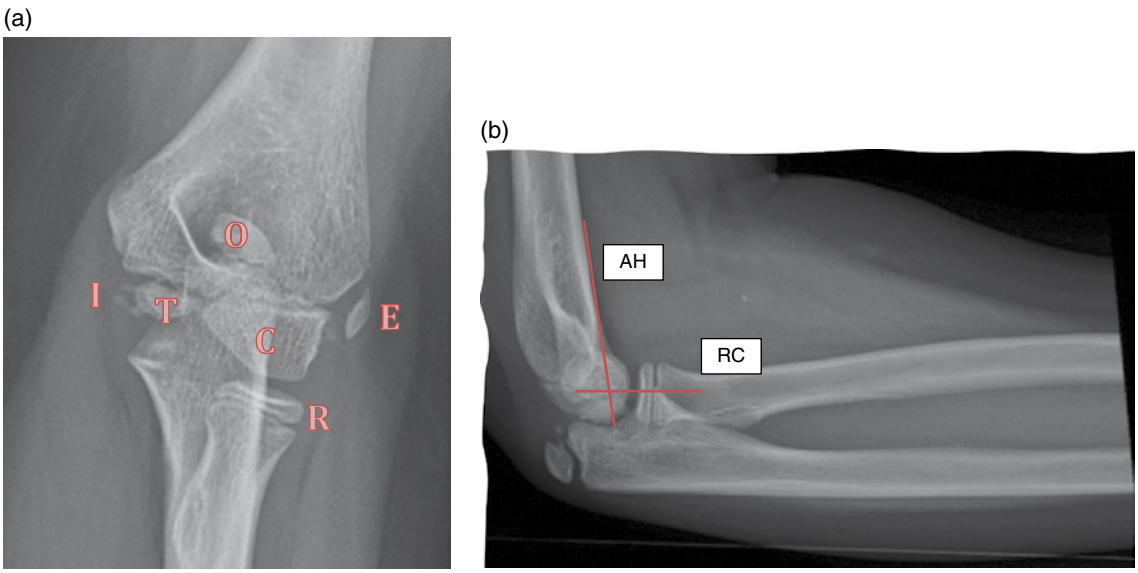

Figure 5.4 (a) Anteroposterior and (b) lateral radiographs of a normal right elbow in a 10-year-old female. (a) Ossification centers of the pediatric elbow appear in order with approximate age: capitellum (C) (1 year), radial head (arrow R) (3 years), internal condyle (I) (5 years), trochlea (T) (7 years), olecranon (O) (9 years), and external condyle (arrow E) (11 years). (b) Note on the lateral view that the anterior humeral (AH) line transects the middle third of the capitellum and that the radio-capitellar (RC) line intersects the middle of the capitellum.

If there is any concern for fracture, the limb should be splinted in a posterior splint and follow-up arranged.

KEY FACT | **A subtle fracture of the elbow can be detected by looking for a posterior fat pad. A *posterior* fat pad of the elbow is always *pathologic*.**

Pitfall | Missing a toddler's fracture in a young child refusing to bear weight and with negative x-rays

The young child with a limp or refusal to bear weight can pose a diagnostic challenge. One specific cause for refusal to bear weight in a child under three years of age is a toddler's fracture. A toddler's fracture is defined as a spiral fracture of the middle or distal tibia that is minimally displaced or nondisplaced. Other considerations in the differential specific for this age are foot foreign body, poorly fitting shoes, infection such as septic arthritis of the hip or knee, transient synovitis, and vaso-occlusive crisis. While most toddler's fractures are associated with trauma, it is often minor and may be dismissed by the parent or caregiver as trivial. If a mechanism is known, a torsional or rotational component is common. Planting of the foot and twisting of the leg or catching of the foot or leg in between the slats of a crib are frequent scenarios for a toddler's fracture.

The clinical examination of a child who refuses to walk should include careful inspection of the thigh, leg, and foot and full range of motion of the hip, knee, and ankle. The most consistent clinical findings in children with toddler's fractures are the inability to bear weight and possibly focal bony tenderness. It is uncommon to find localized swelling or warmth. In fact, most children are quite happy if they are not made to put any weight on the affected extremity. The examiner may be able to elicit pain by placing an axial load on the tibia by compressing the foot toward the knee, or by creating a torsional force on the tibia by stabilizing the knee and rotating the foot. The ability to bear weight and take steps significantly decreases the likelihood of the presence of a toddler's fracture [11].

Radiographs of children with a suspected toddler's fracture can be limited initially to an anteroposterior (AP) and lateral of the tibia and fibula. The AP radiograph is the most likely to reveal the hairline spiral fracture. If this is negative, additional oblique views can increase the likelihood of detection in some cases. Negative radiographs do not rule out this diagnosis. In one study 39% of radiographs were initially negative, but 93% of radiographs obtained at follow-up were diagnostic of a toddler's fracture [12].

A presumptive diagnosis of toddler's fracture should be made in a young child with a history of acute injury, refusal to walk, and absence of signs or symptoms of infection. Treatment of the child with a suspected toddler's fracture includes immobilization in a long leg cast or splint for comfort, to prevent displacement, and to promote healing. Follow-up radiographs at 10–14 days post injury will often show periosteal new bone formation, confirming the diagnosis (Figure 5.6) [13].

KEY FACT | **About 40% of children with a toddler's fracture will have negative initial radiographs. If clinical suspicion is high for toddler's fracture, the child should be placed in a long leg cast or splint and have repeat radiographs performed in two weeks.**

Pitfall | Not considering hip pathology when a young adolescent complains of distal thigh or knee pain

In patients who present with distal thigh or knee pain, pathology of the hips should be considered. Hip pathologies that can cause more distal pain include septic arthritis, transient synovitis, Legg–Calvé–Perthes disease (idiopathic osteonecrosis of the femoral head), an apophyseal avulsion fracture of the hip, and a slipped capital femoral epiphysis (SCFE). SCFE is a frequently missed, time-sensitive pediatric orthopedic problem. Delay in diagnosis leads to worsening displacement of the epiphysis, which can result in worse surgical outcomes and early-onset arthritis [12]. SCFE occurs at the proximal growth plate of the femur. The metaphysis of the femoral neck displaces anteriorly and superiorly in relationship to the femoral head or epiphysis. Most patients will present with a limp and pain in the hip, groin, or proximal thigh. Frequently the pain is chronic, but acute displacement can occur with minor trauma. About 15% of children will have only knee or distal thigh pain at presentation of SCFE [12]. Obesity is a risk factor for SCFE. Typically, displacement occurs during the preadolescent or adolescent growth spurt [12].

A good hip examination is critical to perform in any patient presenting with knee or thigh pain. The most common finding on exam is decreased internal rotation

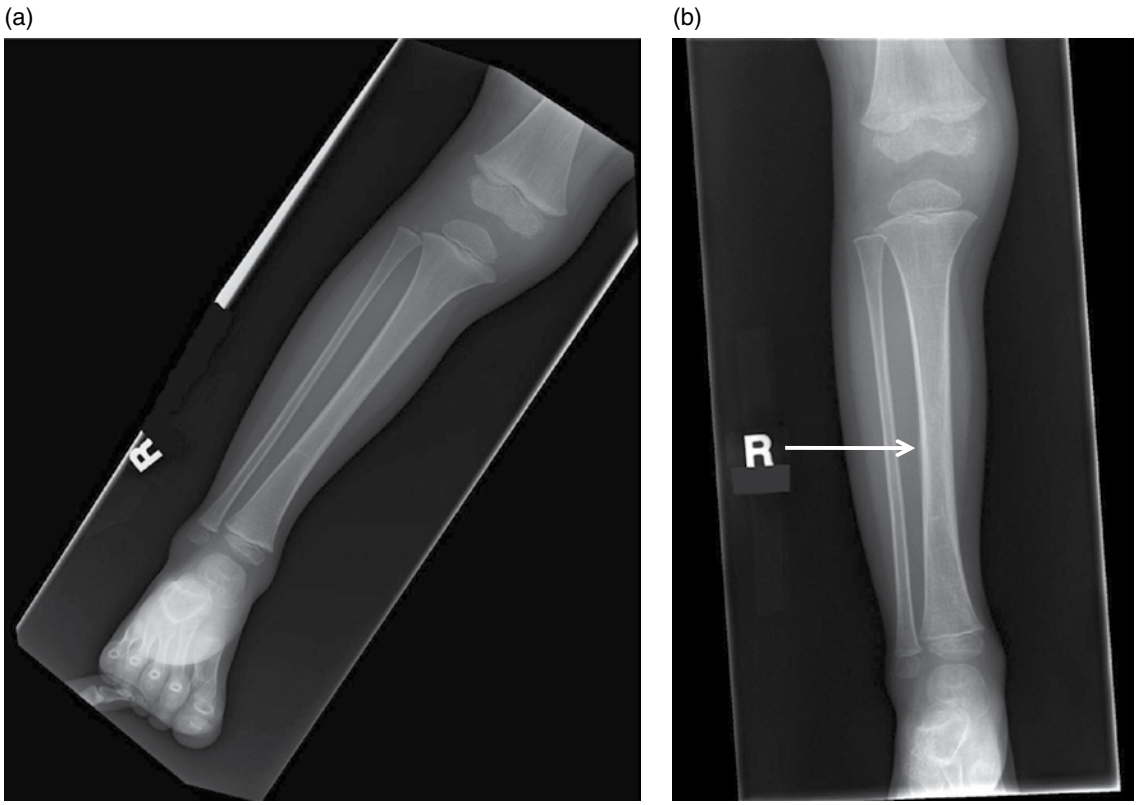

Figure 5.6 Anteroposterior radiograph of tibia/fibula in a two-year-old male. (a) Initial radiograph reveals no visible fracture. (b) Follow-up film at two weeks reveals periosteal reaction and callus formation (arrow), confirming the diagnosis of a toddler's fracture.

of the hip [12]. With hip flexion, frequently there will be obligate external rotation. Diagnosis is made on the AP and frog-leg lateral radiographs of both hips (Figure 5.7). A single AP view of the hips (Figure 5.7(a)) would miss approximately one-third of SCFE cases. Bilateral hip involvement occurs in 20–40% of patients. The AP view will show widening or blurring (Bloomberg's sign) of the proximal femoral growth plate (Figure 5.7(b)). On the AP view, a line drawn along the superior aspect of the femoral neck (Klein's line) should intersect with the lateral portion of the head of the femur. If a slip has occurred, Klein's line will not touch the head of the femur. A 2 mm or greater difference in distance of Klein's line from unaffected side to affected side improves the sensitivity of this measure (modified Klein's line) [13]. The frog-leg lateral view is the best to appreciate displacement of the head of the femur. Once an SCFE has been diagnosed, the child should be made strictly non-weight bearing on crutches. Immediate orthopedic referral is necessary for surgical stabilization and prevention of further displacement.

KEY FACT | **A frog-leg view of the hip should be ordered in addition to an anteroposterior view to diagnose slipped capital femoral epiphysis.**

Pitfall | Not recognizing hip and pelvis apophyseal avulsion injuries in children

An apophysis is a secondary ossification center and is a bony attachment for muscles and tendons. Before they fuse in adolescence, these anatomic variants are commonly mistaken for fractures. There are numerous apophyses in the hip and pelvis that are susceptible to avulsion injury when an eccentric load is applied to the musculotendinous unit that attaches at that location (Figure 5.8). In the pediatric population, the relative

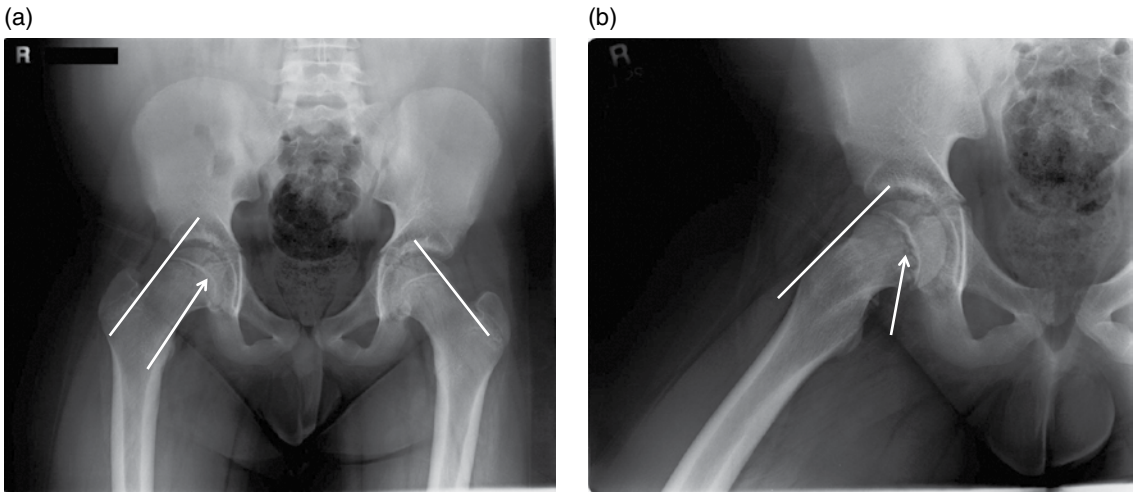

Figure 5.7 (a) Anteroposterior and (b) frog-leg lateral radiographs of the hips in an 11-year-old male with early right slipped capital femoral epiphysis. Klein's line is drawn bilaterally; note that it does not intersect the femoral head on the right side. Note the blurring of the growth plate (arrows; Bloomberg's sign).

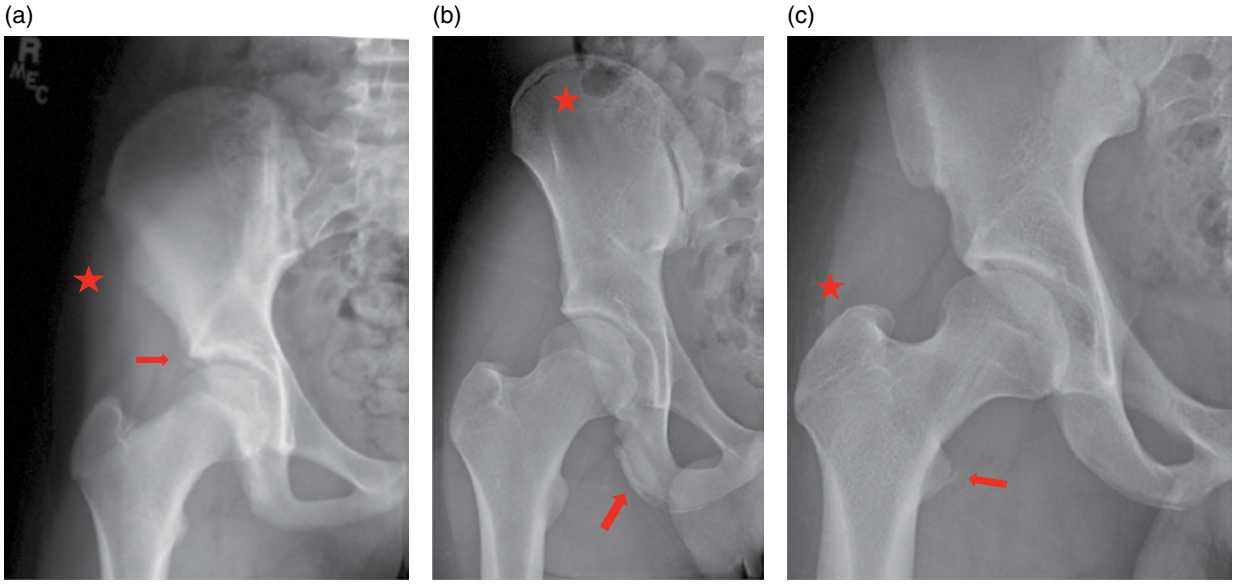

Figure 5.8 Three examples of commonly missed apophyseal avulsion injuries. (a) An anterior inferior iliac spine avulsion injury (arrow) at the attachment of the rectus femoris. Another common location is the anterior superior iliac spine where the sartorius attaches (star). (b) An ischial spine avulsion injury at the attachment of the hamstring tendons (arrow). The star signifies another apophysis at the iliac crest where the abdominal muscles attach. (c) An avulsion injury at the lesser trochanter where the iliopsoas attaches (arrow). The star reflects the location of the greater trochanter where the gluteal muscles attach.

strength of tendons compared to the apophysis results in displacement of the bony apophysis. Unfortunately, x-rays are usually normal in the acute phase; thus clinical suspicion is critical for prompt recognition and treatment.

Apophyseal injuries most commonly occur in adolescence, especially due to sporting activities [14]. Patients will present with acute pain and commonly an antalgic gait. A systematic clinical exam, palpating each apophyseal location and activating the muscles that attach to

that location, can help identify an avulsion fracture. Pain with palpation and pain with active contraction of the attached muscle group are the cornerstones of clinical diagnosis. X-rays should be obtained and scrutinized for displaced bony fragments. Unfortunately, x-rays are commonly read as negative in the acute phase. Management consists of recognition by clinical exam and having the patients transition to protected weight-bearing status on crutches to reduce the potential for further bony displacement. Patients should be referred for specialty follow-up in one week for reassessment and to determine need for further imaging. A structured return to activity and sport is necessary for an optimal outcome. If a bony avulsion is appreciated on x-ray imaging, and is displaced by more than 2 cm, these patients should have follow-up with an orthopedic surgeon within one week for consideration of surgical intervention.

KEY FACT | **Apophyseal avulsion injuries are commonly missed on initial radiographs.**

Pitfall | Failure to consider septic arthritis in a young child with fever and a limp

A septic joint requires rapid identification and treatment to avoid severe and permanent damage. Across all age groups, septic arthritis predominantly occurs in the lower extremities. Septic arthritis occurs more commonly in children than in adults and has devastating consequences when the diagnosis is delayed. The incidence peaks between the ages of two to three years, so when a child presents with fever and limp or refusal to walk, septic arthritis should be the working diagnosis until it is ruled out. If the child appears ill with fever and has a warm, erythematous, painful joint, referral should immediately be made to an emergency department (ED), ideally one with pediatric orthopedic services. However, in the child with fever and a limp who does *not* appear ill, one must be thorough to evaluate for early septic arthritis. Since diagnosis is frequently delayed, septic arthritis of the hip joint is often associated with long-term complications. It is difficult to distinguish between early septic arthritis of the hip and transient synovitis. Transient synovitis is a reactive arthritis of the joint and is generally a diagnosis of exclusion. Unfortunately, both septic arthritis and transient synovitis have many overlapping physical exam findings with the patient resting in their position of comfort, with the hip flexed, abducted, and externally rotated.

On examination, in both septic arthritis and transient synovitis the child will have pain with hip range of motion, particularly with internal rotation. AP and frog-leg lateral radiographs of both hips should be obtained to exclude any bony abnormality. A subtle joint effusion may be appreciated on plain films in both septic arthritis and transient synovitis. Laboratory evaluation is often necessary to stratify the patient for the likelihood of septic arthritis. One cannot rely on leukocytosis to help with diagnosing septic arthritis, as the peripheral white blood cell count is normal in 80% of children with septic arthritis of the hip [15]. Given the complexity of diagnosing septic arthritis of the hip, the Kocher criteria were developed and subsequently validated to aid in this diagnosis (Box 5.1) [16]. Adding the C-reactive protein (CRP) level to the Kocher criteria (the modified Kocher criteria) improves the diagnostic accuracy of using these criteria to diagnose a septic hip. Specifically, a CRP >2 mg/dl is a strong predictor of septic arthritis of the hip [17]. Because the diagnosis is crucial yet difficult to make, when in doubt consider referral to the ED unless there is a structure that allows close follow-up and timely consultations.

KEY FACT | **The peripheral white blood cell count is normal in 80% of children with septic arthritis of the hip.**

Box 5.1 Kocher criteria for septic arthritis of the hip

History of fever >38.5 °C (101.3 °F)	
Non-weight bearing	
Erythrocyte sedimentation rate (ESR) ≥40 mm/h	
White blood cell (WBC) count >12,000 cells/mm^3	
If none of above criteria applies, the risk of septic hip is 2%	
If one criteria applies	10%
If two criteria apply	35%
If three criteria apply	73%
If four criteria apply	93%

Source: Data from Kocher et al. [16].

Pitfall | **Failing to recognize a fracture commonly associated with child abuse**

Encounters for physical abuse among children is not limited to the ED and the urgent care provider should maintain a high index of suspicion for these injuries. The true frequency of fractures related to child abuse is unknown. Estimates suggest that nearly 55% of abused children sustain a fracture at some point in time, second only to soft tissue injuries. It has been found that over 20% of children had at least one previous healthcare visit where the fracture was not recognized as abuse [18]. Young children and developmentally delayed children are at the highest risk for abuse.

When a child presents with a fracture the provider has two goals: to determine whether a fracture is present and if this is the cause of the symptoms, and to determine whether the fracture could be due to child physical abuse. This is a seemingly daunting task; however, the goal of the investigation is simply to gather evidence—it is not to assign guilt.

KEY FACT | **When child abuse is suspected, the role of the healthcare provider is to gather evidence.**

The first step in detecting possible child abuse is being open to the possibility of its existence. Worrisome signs that a fracture is secondary to abuse include delayed presentation to care; inconsistent history; mechanism of injury described that is unlikely to result in the identified injury; multiple fractures; fractures in various stages of healing; and a large amount of callus around a healing fracture possibly due to improper immobilization. Once suspicion is present for abuse, a clinician should initiate the reporting and documentation process. Reporting requirements and procedures are specific to the practice location. The burden of proof does not rest on the shoulders of the clinician, but information gathered early in the evaluation may be critically valuable to social services and child protection agencies.

The American Academy of Pediatrics Council on Child Abuse and Neglect has issued a clinical report detailing the recommended evaluation of suspected child physical abuse [19]. Photo documentation of any cutaneous findings should also be obtained as it is important for future interpretation by a forensic expert. A complete skeletal survey should be considered mandatory in any child under two years of age in whom child abuse is suspected (it does not have to be confirmed) and may be useful in developmentally disabled children up to five years of age who may not be able to provide a history. The skeletal survey helps to identify clinically unsuspected fractures. It is recommended that these radiographs be interpreted by a pediatric radiologist; consequently, referral from the urgent care setting to the ED is often necessary. Infants should undergo retinal examination, by an ophthalmologist, as well as computed tomography (CT) of the head. Liver and pancreatic enzyme testing is performed to evaluate for any potential intra-abdominal injuries. If any abnormalities are detected on physical examination or labs, CT of the abdomen and pelvis should be performed.

While no fracture is truly pathognomonic for child abuse, certain fracture patterns raise the suspicion for inflicted injury. It has been reported that up to 80% of all femur fractures in infants and 40% of all femur fractures in children are the result of abuse (Figure 5.9) [20]. Contrary to common belief, one cannot rely on the type of femur fracture (i.e. spiral, oblique, or transverse) as a way to differentiate accidental from inflicted injury. Over half of children under three years of age with humeral shaft fractures are likely to have been abused [20]. In contrast, supracondylar humeral fractures are almost always the result of accidents.

Skull fractures often raise suspicion for abuse. In children under three years of age, skull fractures are the most common fracture seen in both abusive and nonabusive trauma, with 30% of skull fractures the result of abuse [20]. The type of skull fracture is important. Simple linear skull fractures can be sustained from short falls (<3 ft [<1 m]); however, higher-energy mechanisms result in multiple, comminuted, or diastatic skull fractures and is worrisome for an abusive mechanism, especially when the history reports them as the result of minor trauma.

Rib fractures and metaphyseal fractures (i.e. corner fractures or bucket handle fractures) are the most highly suspicious fractures for abuse. Young children's ribs are cartilaginous and therefore flexible. Fractures of the ribs are the result of extreme force. The probability that a rib fracture is due to abuse is estimated to be over 70% [20].

Corner fractures (also known as bucket handle or metaphyseal fractures) of the distal femur, tibia, and proximal humerus are often due to traction or twisting of the extremity. These can produce minimal pain or deformity and heal without complications and thus may be easy to miss. Early detection can be challenging, and sometimes the fracture only becomes readily apparent

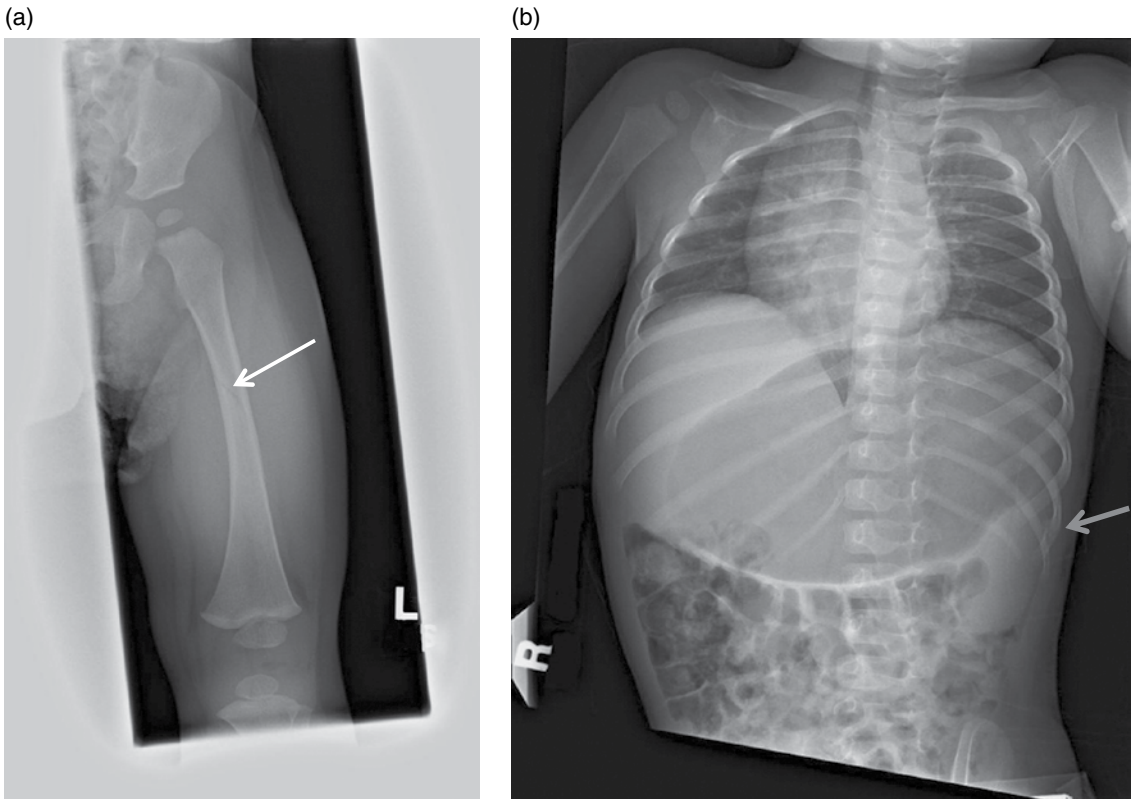

Figure 5.9 Anteroposterior view of left femur and oblique chest radiographs revealing femur fracture and left lateral rib fracture in a 17-month-old infant. (a) Note the nondisplaced oblique fracture of the left femur (arrow). (b) Left lateral rib fracture shows callus formation from a healing previous fracture (arrow).

on follow-up x-rays when callous formation indicates an underlying fracture. In addition, the angle of the x-ray may change the appearance of the fracture. Tangential views will show triangular fracture fragments (corner fracture), whereas the oblique views may show a curvilinear appearance (bucket handle fracture).

Sternum, scapula, and spine fractures are uncommon in children. Injury to these bones requires significant force, such as from a motor vehicle accident. The absence of a clear history of such a mechanism should raise a clinician's concern.

KEY FACT | **While there is no one fracture type that is truly pathognomonic for abuse, one must have a high index of suspicion for abuse when a young child presents with any femur fracture or midshaft humerus fracture.**

Pearls for improving patient outcomes

- Children with open growth plates are more likely to fracture through the growth plate than sustain a sprain.
- Many buckle fractures of the forearm will only be visible on a lateral film.
- Nursemaid's elbow can be diagnosed clinically. If the typical history of being pulled with an outstretched hand without any other trauma is present, a hyperpronation or supination/flexion maneuver to reduce the injury may be performed without radiographs.
- The presence of a posterior fat pad in the setting of traumatic injury to the elbow with otherwise normal radiographs is highly suggestive of occult fracture.
- Normal x-rays are common in toddler's fracture. Immobilization of the extremity should be based on clinical suspicion.
- In a pediatric patient presenting with thigh or knee pain, a hip pathology must be considered.

• Consider apophyseal fractures in pediatric patients with hip and pelvis pain.
• A normal white count does not rule out the possibility of septic arthritis.
• Children frequently complain of knee pain, but this is often referred pain from the hip so the hip should also be examined in any child with knee pain.
• Child abuse should always be a consideration when a child has a fracture or there is bruising in an infant. Rib, metaphyseal, any femur, and midshaft humeral fractures in infants and nonambulatory toddlers represent a high risk of abuse.

References

1 Mann DC, Rajmaira S. Distribution of physeal and nonphyseal fractures in 2,650 long-bone fractures in children aged 0–16 years. *J Pediatr Orthop* 1990; **10**(6): 713–716.
2 Dabash S, Prabhakar G, Potter E, *et al*. Management of growth arrest: current practice and future directions. *J Clin Orthop Trauma* 2018; **9**(Suppl 1): S58–s66.
3 Salter RB, Harris WR. Injuries involving the epiphyseal plate. *J Bone Joint Surg* 1963; **45**(3): 587–622.
4 Singh V, Garg V, Parikh SN. Management of physeal fractures: a review article. *Indian J Orthop* 2021; **55**(3): 525–538.
5 Green A. The pediatric foot and ankle. *Pediatr Clin North Am* 2020; **67**(1): 169–183.
6 Gonzalez N, Lucas J-MP, Winegar A, *et al*. A review of pediatric distal radius buckle fractures and the current understanding of angled buckle fractures. *Cureus* 2022; **14**(5): e24943.
7 Handoll HH, Elliott J, Iheozor-Ejiofor Z, *et al*. Interventions for treating wrist fractures in children. *Cochrane Database Syst Rev* 2018; **12**(12): CD012470.
8 Lee YS, Sohn YD, Oh YT. New, specific ultrasonographic findings for the diagnosis of pulled elbow. *Clin Exp Emerg Med* 2014; **1**(2): 109–113.
9 Krul M, van der Wouden JC, van Suijlekom-Smit LWA, *et al*. Manipulative interventions for reducing pulled elbow in young children. *Cochrane Database Syst Rev* 2012; **1**(1): CD007759.
10 Teach SJ, Schutzman SA. Prospective study of recurrent radial head subluxation. *Arch Pediatr Adolesc Med* 1996; **150**(2): 164–166.
11 Halsey MF, Finzel KC, Carrion WV, *et al*. Toddler's fracture: presumptive diagnosis and treatment. *J Pediatr Orthop* 2001; **21**(2): 152–156.
12 Mathew SE, Larson AN. Natural history of slipped capital femoral epiphysis. *J Pediatr Orthop*. 2019; **39**(1): 23–27.
13 Pattishall AE. An updated approach to toddler fractures. *J Urgent Care Med*. Sept. 1. 2019. https://www.jucm.com/an-updated-approach-to-toddler-fractures/. Accessed July 19, 2024.
14 Choi C, Lee SJ, Choo HJ, *et al*. Avulsion injuries: an update on radiologic findings. *Yeungnam Univ J Med* 2021; **38**(4): 289–307.
15 Laine JC, Kaiser SP, Diab M. High-risk pediatric orthopedic pitfalls. *Emerg Med Clin North Am* 2010; **28**(1): 85–102. viii.
16 Kocher MS, Zurakowski D, Kasser JR. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinical prediction algorithm. *J Bone Joint Surg Am* 1999; **81**(12): 1662–1670.
17 Caird MS, Flynn JM, Leun YL, *et al*. Factors distinguishing septic arthritis from transient synovitis of the hip in children. A prospective study. *J Bone Joint Surg Am* 2006; **88**(6): 1251–1257.
18 Ravichandiran N, Schuh S, Bejuk M, *et al*. Delayed identification of pediatric abuse-related fractures. *Pediatrics* 2010; **125**(1): 60–66.
19 Kellogg ND. Evaluation of suspected child physical abuse. *Pediatrics* 2007; **119**(6): 1232–1241.
20 Kemp AM, Dunstan F, Harrison S, *et al*. Patterns of skeletal fractures in child abuse: systematic review. *BMJ* 2008; **337**: a1518.

CHAPTER 6
Pitfalls of Wound Management

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Introduction

A discussion of wound care is rarely groundbreaking; however, the use of older methods for cleaning and treating wounds can lead to poor outcomes, for both the patient and the provider. This is even more apparent when the practitioner does not have access to specialists to assist with the evaluation and management of complex wounds. Wound care constitutes a large part of the care provided in any acute care facility. Mainly because of the sheer volume of patients with injuries requiring wound care, shortcomings in adequate wound care generate a major amount of litigation. It is estimated that between 5 and 20% of all malpractice claims, and more than 10% of all malpractice dollars, are spent on issues associated with wound care [1].

The key aspects of wound care include cosmetic and functional outcomes, pain relief, and infection control. The key to understanding wound care is realizing that the repair is only the beginning of the treatment. Successful outcomes depend on both the provider and the patient taking their part in the healing process. If the practitioner closes the wound poorly, healing will be less than optimal from the outset. Similarly, if the patient does not comply with post-treatment instructions despite the practitioner's meticulous closure, the wound will not heal suitably.

Wound preparation

Pitfall | **Failure to properly prepare the wound before repair**

There are many factors that influence wound healing; however, no single action is of more importance than proper wound preparation. Prior to closure, proper preparation of the wound can prevent a significant amount of morbidity, namely from infection, that is associated with traumatic wounds. In general, the most important aspects of traumatic wound preparation are appropriate irrigation and debridement of necrotic tissue in the wound. When approaching a traumatic wound the practitioner should first focus on irrigation. The amount of irrigation necessary for the wound is directly related to the expected amount of contamination. In many cases, highly contaminated wounds can require 10 times more irrigation than clean injuries. Another aspect that needs to be taken into consideration is the area where the laceration occurs. Highly vascular areas, for example the face or scalp, require less irrigation to prevent infections. Studies have shown no difference in the rates of infection in clean facial and scalp wounds regardless of the volume of irrigation [2]. In contrast, a foot wound in a diabetic patient necessitates profuse irrigation. The next step in preparing the wound for closure is determining which irrigation solution to use and how it should be applied. Irrigation should be under high pressure and not just soaking. Soaking wounds in iodine solution has shown to be ineffective in preventing infection and it may even promote infection [3]. Although most practitioners believe that the use of sterile solutions is mandatory, irrigation with tap water is equally effective for uncomplicated wounds [4, 5].

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KEY FACT | **Soaking wounds in iodine solution is ineffective in preventing infection and it may even promote infection.**

What seem to be most important are the amount and pressure with which the wound is irrigated. The most effective technique is to irrigate at pressures of 5–8 psi. To do this use a 20–60 ml syringe attached to a 19–20G needle or catheter [4]. Overzealous irrigation at pressures higher than necessary may result in further damage to the tissue. Take care not to apply too much pressure to areas with delicate microvasculature, such as the eye and ear. Overaggressive irrigation in these areas can separate the overlying tissue from the cartilage and cause necrosis [6]. A volume of 50–100 ml/cm of wound is a good place to begin. The use of tissue-toxic solutions should be avoided. These include povidone–iodine solutions and hydrogen peroxide. These solutions can devitalize the healthy tissue and provide a medium for bacteria to flourish, thus impairing wound healing [7]. There is some evidence to suggest that the use of a 1% povidone–iodine solution in infection-prone wounds is beneficial [8, 9]. This, however, is controversial because this antiseptic, especially in higher concentration, impairs wound healing.

KEY FACT | **Wounds should be irrigated with 50–100 ml/cm of wound.**

Foreign bodies

Pitfall | Failure to inform the patient about the possibility of a foreign body

On the initial evaluation of a patient, one of the key aspects of the history that must be obtained is the possibility of the presence of a foreign body. There are a few aspects of the history that should alert the practitioner to be more aggressive in the evaluation for a foreign body. These include injuries caused by glass, foreign-body sensation, or persistent pain and drainage [10]. After a thorough history, the physical examination can help to elucidate, but not eliminate, the possibility of a retained foreign body. The most ideal situation would be to examine all wounds in the absence of blood and through the entire length of the wound. This would seem to eliminate any chance of a foreign body; however, even with direct visualization of the base of the wound there remains an almost 20% chance of a retained foreign body [1].

Any injury that was caused by, or affected by, glass should be evaluated with an imaging modality, since the most likely retained foreign body is glass. In the past, the use of plain films was the only way to evaluate for foreign bodies. Although the use of plain radiography does further reduce the possibility of a retained foreign body, it does have some limitations. Wounds that retain vegetative matter, for example wood or thorns, may not have any noticeable abnormality on films. It must also be considered that some forms of plastic are radiolucent and will be very difficult or impossible to visualize. The unfortunate paradox is that wounds caused by these objects have a higher likelihood of becoming infected, which places the practitioner in a difficult situation. The use of multiple views and markers (e.g. paper clips) can be helpful in determining the location of foreign bodies in three dimensions when using plain films.

KEY FACT | **Even with direct visualization of the base of the entire wound, there remains an up to 20% chance of a retained foreign body.**

If your facility is fortunate enough to have other imaging modalities, these can be extremely useful in the detection of foreign bodies. Most hospital-based facilities will have computed tomography (CT) scanning and ultrasound services, in addition to plain films. CT scanning has two main advantages over plain films in that it is 100 times more sensitive for the detection of a foreign body and it also allows for three-dimensional localization of the object [11]. The use of ultrasound to visualize retained objects has a unique benefit when compared to other modalities in that it allows for "real-time" visualization. In essence, you can search for the object with forceps or a needle and determine your location when compared with the object. Additionally, plant material and plastics are not well visualized with x-ray or CT imaging but are readily visualized with ultrasound. Since smaller body parts such as digits or palms/soles are frequently involved in cases of retained foreign bodies, optimal visualization can be achieved with use of a water bath or spacer to enhance specificity. Use of a high-frequency or "small parts" ultrasound probe is also recommended [12]. Both CT scanning and ultrasound use may not be readily available and the

patient may ultimately need referral for further management.

One important fact to remember is that injuries are not the only causes of retained foreign bodies. Occasionally the repair of a wound can necessitate the use of deep sutures to facilitate closing. These deep sutures are, in essence, foreign bodies and will provoke an immune response. Deep sutures should only be used when they are absolutely necessary and should be limited to areas that are less likely to become infected.

Plantar wounds

Pitfall | **Failure to appreciate the high-risk nature of plantar puncture wounds**

Plantar wounds provide a difficult situation for the acute care practitioner. Patients do not always present immediately after the injury since bleeding is not usually a major concern from these injuries. In addition to the delayed time from injury to presentation, there are other factors that influence the rate of infection. The three main factors that influence the propensity for plantar wounds to become infected are the mechanism of injury, the inherently contaminated area of the foot, and the structural anatomy. A particular concern for the practitioner is the fact that most plantar wounds are punctures. Unlike a laceration that is formed by a sweeping motion of a sharp object, a puncture wound is formed by pushing perpendicular to the epidermis. This force acts to push foreign bodies into the wound and trap them there. In addition, the skin may actually close at the surface depending on the size of the object causing the injury, effectively sealing the contaminants into the wound. While the mechanism is important to understand the risk of infection, it is also imperative to understand the bacterial flora present in the plantar area and how this can change with different footwear. As we walk, our feet are in constant contact with the surrounding environment. This interaction provides countless opportunities for wounds to occur on the plantar surface. Overall gram-positive bacteria are the most common bacteria found in plantar wound infections; however, when the injury involves rubber-soled shoes *Pseudomonas* infection must be considered [10]. Therefore, when the wound occurs through rubber-soled shoes, the use of an anti-*Pseudomonal* agent should be considered as first-line therapy. Unfortunately, the only current recommendation for oral treatment is fluoroquinolones, which have a significant side effects.

KEY FACT | **When the wound occurs through rubber-soled shoes, the use of ciprofloxacin should be considered first-line therapy.**

The final characteristic of plantar wounds that contributes to their morbidity is the structural anatomy of the foot. The proximity of the skin to important structures such as joints, in conjunction with the force exerted onto the foot from the weight of the individual, contributes to high rates of infection, including osteomyelitis. Additionally, the fact that the feet are the farthest body part from the central circulation also contributes to the increased propensity for infection because of lesser blood flow.

Treatment for plantar punctures is generally the same for all other types of wounds. There should be a thorough search for retained foreign bodies, which can include plain radiographs or referral for more sensitive tests. Although the wound should be cleansed using high pressure (5–8 psi) with a nontoxic solution, one cannot simply place the needle into the puncture wound as this may force contaminants into the wound. If the wound is small and prevents adequate irrigation, the puncture wound should be converted into a laceration to aid in proper irrigation. Wounds that are grossly infected can be "cored out," leaving a large area for drainage of the infection [13]. Finally, as mentioned earlier, the judicious use of antibiotics is indicated for these wounds, keeping in mind the prevailing organisms.

Wound care instructions

Pitfall | **Failure to provide appropriate wound care and follow-up instructions**

One of the most important and often overlooked aspects of wound care is the post-discharge set of instructions. The time spent with the practitioner is minuscule in comparison to the total time the wound needs to heal. Proper instructions can eliminate some of the long-term complications that are inherent with traumatic wounds. These instructions should be succinct, easily understandable, and most importantly provide the patient with reasonable expectations of the healing process. One of the main concerns for patients presenting for

wound care is cosmesis, or the visual appearance of the healed wound [14]. It is important to explain the healing process of the wound, especially that all wounds leave scars caused by the healing process. Patients should also be recommended to avoid ultraviolet light exposure during the wound-healing process, as this may produce permanent hyperpigmentation of the wound.

In an effort to save time and promote understanding the instructions can be given twice, with the first being during the closure of the wound. The patient at this point is a captive audience and this allows the practitioner to have their full attention. The second set of instructions comes prior to discharge with more formal written instructions that the patient can keep for future reference. The instructions should discuss cleaning, bandages, antibiotic ointments, and follow-up. Cleaning closed wounds is always advised. As discussed previously, harsh tissue-toxic materials like hydrogen peroxide should not be used. The wounds can be cleansed with mild soap and water and then dried carefully, usually by patting dry. This can happen as early as four to six hours after closure. Drying the wound is an especially important point to convey, since wounds that are excessively wet can cause the skin at the edges of the wound to degrade and ultimately lead to dehiscence of the wound. The choice of bandage should be guided by the location of the wound. The bandage should protect the wound from contamination, trauma, and sunlight while allowing for a proper environment for wound healing.

If the patient is expected to change the dressings, explicit instructions on dressing changes must be provided. The use of bacitracin is very common but current data suggests that it is a common cause of contact dermatitis and may impede wound healing [15]. Over-the-counter petroleum-based antibiotic ointments are effective and there is little to no disadvantage to their use [16]. A petroleum-based ointment should not be used if skin adhesive was employed to close the wound, as they can prematurely dissolve the adhesive.

The final aspect of the information should provide return instructions. Most wounds should be reevaluated by a healthcare provider within 48 hours, although this is just a guideline and remains highly flexible and patient dependent. Highly contaminated wounds or bites may need to be examined within 24 hours. Conversely, clean wounds in a reliable patient may not need to be seen until the removal of sutures. The patient should be instructed to return at the first sign of infection, even if it is before the scheduled follow-up visit. Finally, the patient should be given clear instructions on when to have the sutures or staples removed.

Zipper injuries

Pitfall | **Failure to appropriately manage zipper injuries**

Although not very common, zipper injuries can be both emotionally and physically painful to the patient. Due to the anatomy of the region, boys tend to be affected more frequently than girls. If the skin is entrapped within the sliding mechanism the extrication can be challenging. An acute care provider will be tempted to release the entrapped skin by forcibly unzipping. This procedure can result in repeated trauma and should be reserved as the last resort.

The technique for extrication is based on the location of the skin in relation to the sliding mechanism. If the skin is entrapped between the teeth of the zipper, a provider can cut the cloth around the affected area and gently pull apart the teeth of the zipper (Figure 6.1). The most common method involves using wire or bone cutters to cut the median bar, separate the anterior and posterior faceplates, and release the skin. The median bar may be quite strong and will require a considerable amount of force when severing it. Another option is to soak the affected skin in mineral oil for approximately 15 minutes before gently pulling the skin away from the teeth [18].

Another reported mechanism for removing the faceplate is to insert a small flathead screwdriver between the faceplate and rotate it toward the median bar to release the skin (Figure 6.2).

There is no single superior method to treat a zipper injury [20]. Each case must be treated individually and the appropriate technique applied. Besides considering the suitable technique, the provider should not overlook the importance of analgesia and sedation, especially in pediatric patients. The weight of the pants can cause more pain and trauma, thus the provider must work swiftly to cut the clothes around the injury to release tension and gain access to the area. Cases in younger children may require general anesthesia and possible circumcision. Emergent consultation is required in these cases.

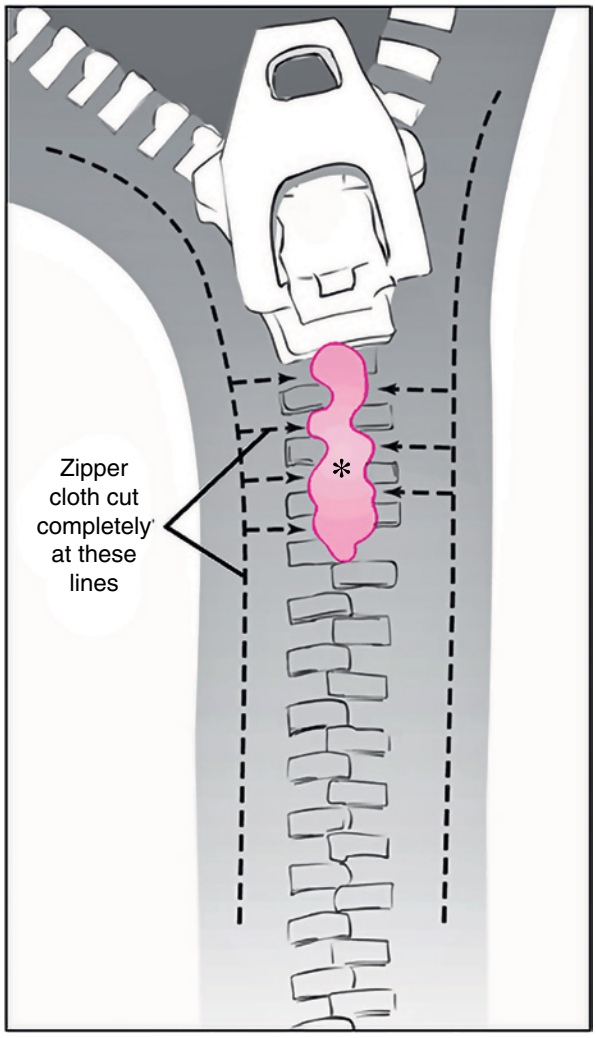

Figure 6.1 Cut below the entrapped skin (asterisk) and pull the teeth of the zipper apart to release it. *Source*: Reproduced with permission from Fein and Zderic [17]/Wolters Kluwer Health, Inc.

Burn management

Pitfall | **Overlooking burns to high-risk areas**

Acute care providers will regularly treat small and superficial burns [21]. Burned tissue should be cleansed with mild soap and water, assessed for debridement, and dressed appropriately. Debridement of devitalized tissue, for example from a ruptured blister of a superficial partial-thickness burn, is important to prevent infection. However, there is mixed evidence regarding rupturing intact blisters. Large (>6 mm in diameter) thin-walled blisters with unclear underlying wound-bed depth should be ruptured and debrided [22]. Historically, silver sulfadiazine (SSD) was used for most burns due to its antimicrobial properties. However, it has fallen out of favor recently as it has been shown to delay wound healing and to be associated with other adverse effects. Alternative dressing agents to consider include honey, antibiotic ointment, or petroleum-impregnated gauze, covered by a dry dressing. It is worth noting that bacitracin- and Polysporin®-containing ointment preparations may result in contact dermatitis in some patients, and petroleum-impregnated gauze lacks antimicrobial properties. Alternatively, hydrocolloid or silver-impregnated foam dressings can be used if available [23].

Providers must be aware of the types of burns that may be at high risk for infection, disfigurement, and loss of function. For example, the treatment of facial burns requires special consideration. SSD is contraindicated in facial burns because of a skin-bleaching effect. Additionally, when the burn involves the eye the provider needs to carefully assess the eye and surrounding structures. The injured area requires copious irrigation to remove debris and cool the surface of the cornea. Burns around the eyes and cornea are treated with topical ophthalmic antibiotic ointment and a generous application of artificial tears. Any burns to the eyelid should raise alarm, since they may result in permanent contractures and an inability to close the eyelid. Thus patients with these burns should be referred to a burn center for further management. Other special populations in which SSD is contraindicated include pregnant and breastfeeding patients, as well as infants because of the risk of causing kernicterus in infants.

KEY FACT | **Silver sulfadiazine should be avoided in pregnant women and nursing mothers since it may cause kernicterus in infants.**

Burns to the hands pose many challenges on initial presentation. The provider must assess the vascular integrity as these injuries are at high risk for compartment syndrome. If there is concern for compromised blood flow to the hands and fingers, the patient should be referred promptly to a burn center for further management as an emergency escharotomy or fasciotomy may be required. The burned hand should be splinted in a position of function and elevated to decrease edema. Each digit

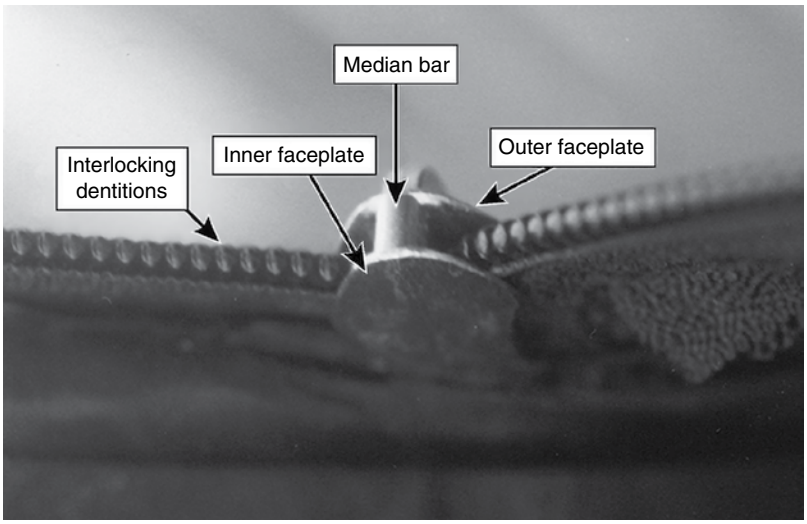

Figure 6.2 Anatomy of a zipper's sliding mechanism. *Source*: Reproduced with permission from Raveenthiran [19]/ Wolters Kluwer Health, Inc.

should be dressed separately to prevent adherence to one another. Early mobility and rehabilitation are often recommended to preserve function and decrease the risk of long-term contractures [24]. Extensive burns to the face, hands, feet, and perineum—as well as patients who meet the criteria for a major burn—should be transferred to a burn center [22].

In pediatric cases, child abuse should be considered whenever a toddler presents with delineated and symmetric burns to the buttock or thighs, which are suggestive of scalding by immersion. A deep circular wound should raise suspicion of a cigarette burn. If there are any concerns, prompt referral to child protective services is warranted.

Depending on the location of your practice, sunburn may represent a large portion of your burned patients. Fortunately, the short-term treatment of these injuries is mainly supportive. The key to proper sunburn treatment lies in the prevention of the injury with proper sun protection. The patient will present with erythema, warmth, tenderness, and even blistering in severe cases. If the patient is presenting for treatment, analgesia with nonsteroidal anti-inflammatory drugs or acetaminophen is appropriate. The use of aloe and steroids, both systemic and topical, is controversial. These agents may help with symptoms but do not shorten recovery time, and the use of steroids can increase the risk of systemic infection in severe burns [25].

Pearls for improving patient outcomes

- The most important aspects of wound preparation are appropriate irrigation and debridement of necrotic tissue in the wound.
- Evaluate every wound for the presence of foreign bodies and inform the patient about the possibility of their existence.
- Beware the high risk of infection in patients with plantar puncture wounds.
- Provide appropriate analgesia when treating patients with zipper injuries.
- Maintain a low threshold for transferring patients with hand burns to burn centers.

References

1 Pfaff J, Moore G. ED wound management; identifying and reducing risk. *ED Leg Lett* 2005; **16**: 97–108.
2 Hollander JE, Singer AJ. Laceration management. *Ann Emerg Med* 1999; **34**: 356–367.
3 Chisholm CD. Wound evaluation and cleansing. *Emerg Med Clin North Am* 1992; **10**: 665–672.
4 Capellan O, Hollander JE. Management of lacerations in the emergency department. *Emerg Med Clin North Am* 2003; **21**: 205–231.
5 Bansal BC, Wiebe RA, Perkins SD, *et al*. Tap water for irrigation of lacerations. *Am J Emerg Med* 2002; **20**: 469–472.
6 Brown DJ, Jaffe JE, Henson JK. Advanced laceration management. *Emerg Med Clin North Am* 2007; **25**: 83–99.

7 Oberg MS, Lindsey D. Do not put hydrogen peroxide or povidone iodine into wounds! *Am J Dis Child* 1987; **141**: 27–28.

8 Oberg MS. Povidone-iodine solutions in traumatic wound preparation. *Am J Emerg Med* 1987; **5**: 553–555.

9 Dire DJ, Welsh AP. A comparison of wound irrigation solutions used in the emergency department. *Ann Emerg Med* 1990; **19**: 704–708.

10 Moayedi S, Torres M. Wound care in emergency medicine. In: Mattu A, Goyal D (eds.), *Emergency Medicine: Avoiding the Pitfalls and Improving the Outcomes*. Malden (MA): Blackwell; 2007: pp. 72–78.

11 Bauer AR Jr, Yutani D. Computed tomographic localization of wooden foreign bodies in children's extremities. *Arch Surg* 1983; **118**: 1084–1086.

12 Dean AJ, Gronczewski CA, Costantino TG. Technique for emergency medicine bedside ultrasound identification of a radiolucent foreign body. *J Emerg Med* 2003; **24**: 303–308.

13 Lammers RL. Principles of wound management. In: Roberts JR, Hedges JR (eds.), *Roberts and Hedges' Clinical Procedures in Emergency Medicine* (5th edn.). Philadelphia (PA): Saunders Elsevier; 2009: p. 565.

14 Singer AJ, Mach C, Thode HC Jr, *et al*. Patient priorities with traumatic lacerations. *Am J Emerg Med* 2000; **18**: 683–686.

15 Saik RP, Walz CA, Rhoads JE. Evaluation of a bacitracin-neomycin surgical skin preparation. *Am J Surg* 1971; **121**: 557–560.

16 Smack DP, Harrington AC, Dunn C, *et al*. Infection and allergy incidence in ambulatory surgery patients using white petrolatum vs bacitracin ointment. A randomized controlled trial. *JAMA* 1996; **276**: 972–977.

17 Fein JA, Zderic SA. Management of zipper injuries. In: King C, Henretig FM (eds.), *Textbook of Pediatric Emergency Procedures* (vol. **2e**). Philadelphia (PA): Lippincott, Williams & Wilkins; 2008: p. 908.

18 Raveenthiran V. Releasing of zipper-entrapped foreskin: a novel nonsurgical technique. *Pediatr Emerg Care* 2007; **23**: 463.

19 Kanegaye JT, Schonfeld N. Penile zipper entrapment: a simple and less threatening approach using mineral oil. *Pediatr Emerg Care* 1993; **9**: 90–91.

20 Inoue N, Crook SC, Yamamoto LG. Comparing 2 methods of emergent zipper release. *Am J Emerg Med* 2005; **23**: 480–482.

21 Singer AJ, Dagum AB. Current management of acute cutaneous wounds. *N Engl J Med* 2008; **359**: 1037–1046.

22 Sargent RL. Management of blisters in the partial-thickness burn: an integrative research review. *J Burn Care Res* 2006; **27**(1): 66–81.

23 Norman G, Christie J, Liu Z, *et al*. Antiseptics for burns. *Cochrane Database Syst Rev* 2017; **7**(7): CD011821.

24 McKee DM. Acute management of burn injuries to the hand and upper extremity. *J Hand Surg Am* 2010; **35**: 1542–1544.

25 Lowe NJ. An overview of ultraviolet radiation, sunscreens, and photo-induced dermatoses. *Dermatol Clin* 2006; **24**: 9–17.

CHAPTER 7
Emergency Dermatology for the Acute Care Provider

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Introduction

Dermatologic problems account for nearly 4.5 million emergency department (ED) visits annually, representing 3.2% of all ED visits [1]. While most dermatologic problems are benign, skin problems can represent cutaneous manifestations of severe underlying medical disease. Such entities require early recognition by the acute care provider to ensure prompt treatment in a setting matching the patient's acuity.

The primary objective of this chapter is to outline a rational approach to the patient who presents with a rash in an urgent care setting. Figure 7.1 outlines this in detail. Three questions will be central to this approach: (1) Does the patient with a rash have findings and features suggestive of serious systemic medical disease or toxicity (i.e. a dermatologic emergency)? (2) Does the patient have a common, clinically identifiable disease for which treatment can be initiated? (3) Does the patient have a problem for which corticosteroid therapy—topical or oral—will be beneficial? Using these questions, the acute care provider can more efficiently, safely, and effectively manage patients who present with a rash.

Dermatologic emergencies

Pitfall | **Failure to recognize a rash as a manifestation of a life-threatening underlying condition, constituting a dermatologic emergency**

The first objective in evaluating any patient with a rash is to identify signs, symptoms, or clinical features that are suggestive of a life-threatening dermatologic emergency. These are detailed in Box 7.1. A full set of vital signs must be obtained on any patient presenting with a rash. Vital sign abnormalities, rashes with extensive blistering, skin sloughing or mucosal involvement, and the presence of petechiae all increase the likelihood that the patient has a dermatologic emergency. Patients with dermatologic emergencies presenting to an urgent care setting will require prompt transfer to a higher level of care and early initiation of treatment to prevent morbidity and mortality. Five dermatologic emergencies will be discussed in detail in this section: Rocky Mountain spotted fever (RMSF), meningococcemia, Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), staphylococcal toxic shock syndrome (STSS), and necrotizing fasciitis. These entities exemplify the findings and features suggestive of serious underlying disease in the patient presenting with a rash.

Pitfall | **Failure to identify a petechial rash**

Petechiae are small (1–2 mm) red or purple lesions that result from hemorrhages under the skin. They are usually flat and do not blanch when pressed. It can be easy to misidentify a nonblanching rash unless one specifically looks for blanching, which is best appreciated by using a clear glass to apply pressure to the skin and observing whether the petechiae fade with pressure. While petechial rashes may be benign secondary to

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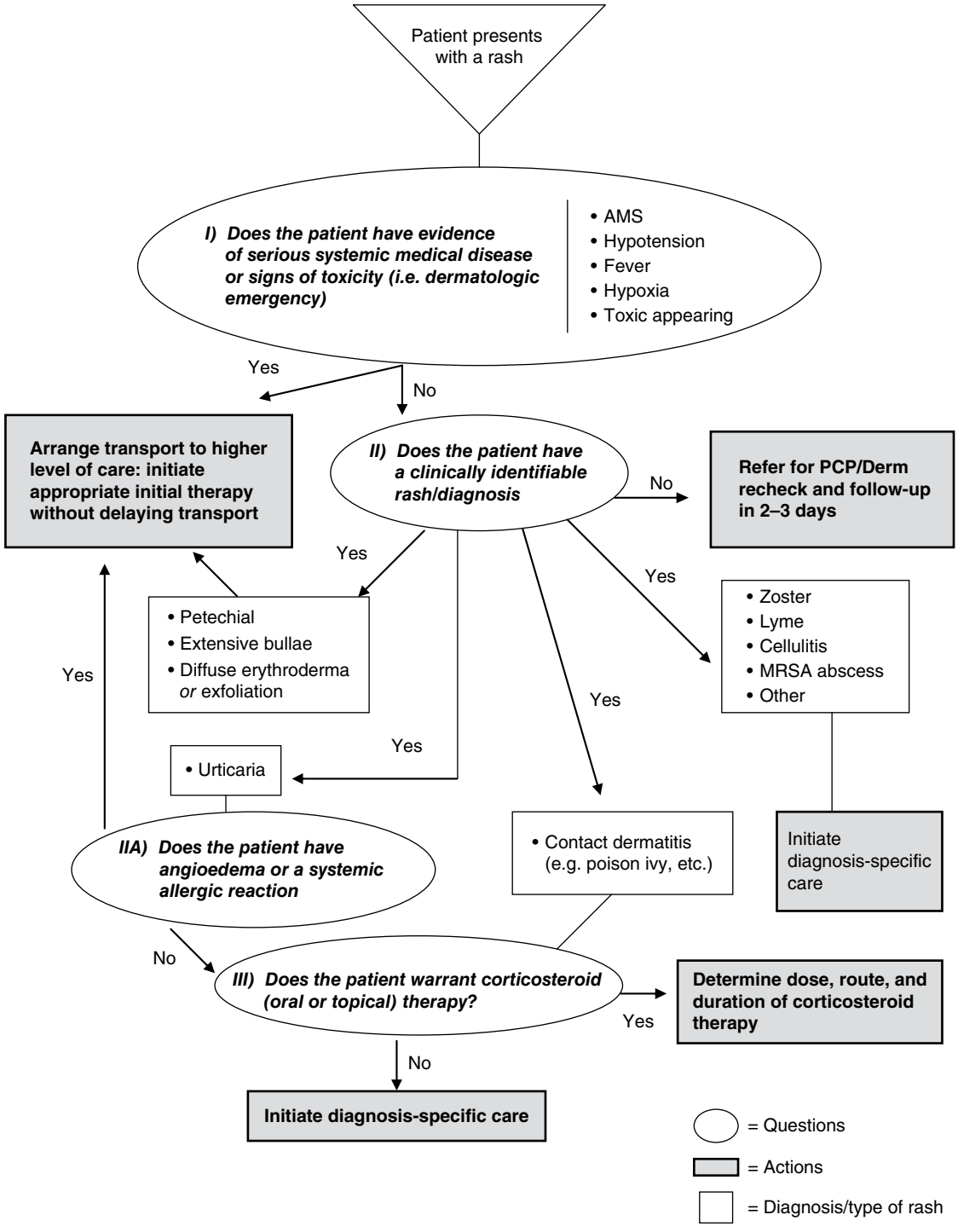

Figure 7.1 Approach to the patient who presents with a rash. AMS, altered mental status; MRSA, methicillin-resistant *Staphylococcus aureus*; PCP, primary care physician.

Box 7.1 Signs, symptoms, and clinical features suggestive of a dermatologic emergency

- Hypotension
- Tachycardia
- Hypoxia
- Fever >38.9 °C (102 °F)
- Pain out of proportion to clinical findings
- Toxicity (ill appearance)
- Altered mental status (AMS)
- Dyspnea
- Mucosal involvement (genital, oral, or conjunctival)
- Extensive blistering, skin sloughing, or desquamation
- Petechiae
- Meningeal signs (Kernig and Brudzinski signs)

trauma or viral infection, they should heighten suspicion for serious conditions including RMSF and meningococcemia. The acute care provider should assume a life-threatening cause of petechiae until proven otherwise.

Rocky Mountain spotted fever

RMSF is a tickborne illness caused by *Rickettsia rickettsii*, a small Gram-negative bacterium. It is spread by several species of ticks in the United States, including the American dog tick (*Dermacentor variabilis*), Rocky Mountain wood tick (*Dermacentor andersoni*), and, in parts of the southwestern United States and Mexico, the brown dog tick (*Rhipicephalus sanguineus*). Despite its name, most (>60%) cases occur in North Carolina, Tennessee, Missouri, Arkansas, and Oklahoma [2]. Most cases occur between May and August [2]. People with the highest likelihood of exposure to ticks are at the greatest risk.

Classic clinical presentation is fever, headache, and rash in a person with a history of tick exposure. Severe abdominal pain is common in children. While most (90%) people with RMSF develop some type of rash during the course of illness, <50% have a rash during the first three days of illness, when patients first seek medical care. The rash usually appears two to four days after the onset of fever in 80–90% of patients [3]. It begins around the wrists and ankles as small, blanching, nonpruritic macules that progress to nonblanching petechial lesions involving the palms, soles, trunk, and extremities, with the face usually spared. Diagnosing RMSF is challenging; 60–75% of patients with this condition are initially given an alternate diagnosis [4], which can lead to poor outcomes as morbidity has been shown to be as high as 35% when treatment is delayed beyond day 5 of illness [5]. As such, antimicrobial therapy should be initiated as soon as possible in all patients suspected to have RMSF. Doxycycline 100 mg orally twice a day for 7–10 days is first-line therapy. Patients with mild disease who appear well can generally be treated as outpatients. Patients with severe symptoms should be hospitalized, as complications include shock, acute renal failure, seizures, coma, hepatic failure, and death. In these critically ill patients, administer an initial intravenous (IV) loading dose of 200 mg doxycycline.

KEY FACT | **Rocky Mountain spotted fever is associated with a 4–9% mortality among treated patients and a 30–70% mortality among those not treated promptly or adequately.**

Meningococcemia

Neisseria meningitides, a Gram-negative diplococcus bacterium, is a leading cause of septicemia and meningitis among small children and young adults in the United States and is associated with high mortality [6]. The clinical presentation of invasive meningococcal disease is variable. Classically, the patient presents with sudden onset of prodromal fever, malaise, nausea, vomiting, headache, and a rash [7]. A maculopapular eruption resembling a wide variety of viral exanthems can be an early finding of meningococcemia; however, this rash is transient and generally does not persist for more than two days. Following this early eruption, a petechial rash, which is the pathognomonic feature of meningococcemia, develops in 50–60% of cases and must be promptly identified by the acute care provider. This rash appears as discrete lesions 1–2 mm in diameter starting on the wrists, ankles, and axillae, and spreading to the trunk and extremities [8].

A thorough physical exam should be performed with the patient fully undressed, as sites where pressure is applied to the skin will likely reveal petechiae (e.g. under elastic straps and belts). Head, palms, and soles are often spared. Petechiae can coalesce into larger purpuric lesions.

If unrecognized or untreated, the disease can rapidly progress to shock, disseminated intravascular coagulation, acute renal failure, and death. Early recognition, prompt

administration of appropriate antibiotics, treatment of shock, and transfer to a facility with intensive care capabilities are critical.

Stevens-Johnson syndrome and toxic epidermal necrolysis

Pitfall | Failure to examine a patient with a rash for mucosal involvement

SJS and TEN are conditions that represent a spectrum of drug-induced or idiopathic mucocutaneous reaction patterns. Roughly 50% of SJS and 80–90% of TEN patients are drug-induced, with the most commonly implicated agents being sulfa drugs, anticonvulsants, allopurinol, antibiotics, and nonsteroidal anti-inflammatory drugs (NSAIDs) [1, 9]. Both are characterized by viral prodrome (fever, malaise, myalgias, sore throat, conjunctivitis) followed by development of a macular exanthema that begins centrally then spreads to the extremities. Atypical target lesions with central dusky purpura can be seen [10]. The exanthem eventually becomes confluent and then converts to blistering with dermal–epidermal dissociation and sloughing. This denudation, which occurs with shear stress, is termed a "positive Nikolsky's sign." One of the earliest signs is mucosal involvement, with up to 90% of patients having mucosal lesions such as painful erosions or surface crusting [11]. This can include oral cavitary (90% of patients), ocular (84%), genital (60%), nasal (50%), ear (50%), and laryngeal (50%) involvement [12–14]. Early identification requires careful examination of these areas.

KEY FACT | **The clinical hallmark of Stevens-Johnson syndrome and toxic epidermal necrolysis is mucous membrane involvement with erythema and erosions of oral, genital, and ocular mucosa.**

TEN and SJS are differentiated by the percentage of the body surface area (BSA) involved, with <10% defined as SJS, 5–30% defined as SJS/TEN, and >30% defined as TEN [10]. Epithelial sloughing results in susceptibility to bacterial infection, septicemia, and significant fluid loss. Mortality varies from 12 to 49% across the SJS/TEN severity spectrum [15]. These patients will need supportive care that includes IV fluids, corticosteroids, antimicrobials, and potentially burn unit care if there is extensive BSA involved.

Staphylococcal toxic shock syndrome

Pitfall | Failure to elicit a history of a foreign body (e.g. tampon, wound packing) in a patient presenting with a diffuse rash and fever

STSS is a multisystem, fulminant illness induced by toxins produced by various strains of *Staphylococcus aureus* bacteria. It is most often observed in menstruating women using highly absorbent tampons or in patients with nasal packing [1]. After a two- to three-day prodrome of malaise, patients typically present with fever, chills, nausea, abdominal pain, and hypotension. The rash of STSS typically develops in the first 48 hours of the illness. It is a diffuse, red, macular rash resembling sunburn that can involve the palms and soles and may have a "sandpaper" quality on palpation. Desquamation of the skin generally occurs one to two weeks following the onset of symptoms [16].

Six criteria must be met for the diagnosis of STSS: (1) fever ≥38.9 °C (102°F); (2) diffuse macular erythroderma; (3) desquamation one to two weeks after onset of rash; (4) hypotension (systolic blood pressure ≤90 mmHg); (5) evidence for the absence of other illness, such as sepsis; and (6) clinical or laboratory findings in at least three other organ systems: gastrointestinal, renal, hematologic, neurologic, hepatic, musculoskeletal, or mucosal membrane. Treatment is prompt removal of the inciting factor (i.e. tampon), fluid resuscitation, treatment of shock, and broad-spectrum antibiotics to include IV vancomycin *plus* IV clindamycin (suppresses toxin synthesis) *plus* a combination drug containing a penicillin plus a beta-lactamase inhibitor such as IV piperacillin-tazobactam. Identification and removal of the foreign body is critical in these patients because antibiotics alone will be insufficient if the nidus of infection remains.

Necrotizing soft tissue infections

Pitfall | Failure to recognize pain out of proportion to physical findings, thereby errantly diagnosing a necrotizing soft tissue infection as simple cellulitis

Necrotizing soft tissue infections (NSTIs), including necrotizing forms of fasciitis, myositis, clostridial myonecrosis, and Fournier gangrene, are a dangerous subset

of skin and soft tissue infections characterized by fulminant tissue destruction, systemic signs of toxicity, and high morbidity and mortality [17]. Risk factors for developing NSTI include immunosuppression, trauma, diabetes, peripheral vascular disease, alcohol or IV drug use, malnutrition, and obesity. NSTI can be caused by multiple synergistic bacteria (Type I) or a single organism (Type II) such as group A beta-hemolytic streptococcus. The most common sites are the extremities and perineum (Fournier gangrene). Clinical manifestations include erythema, severe pain, soft tissue edema, fever, and skin bullae or necrosis [18, 19]. Presence of soft tissue infection with signs of systemic illness, in addition to pain out of proportion to clinical examination findings and the presence of crepitus (35% of cases), differentiates necrotizing fasciitis from simple cellulitis [18, 19]. Such severe pain should not be attributed to drugs or attention-seeking behavior. Although NSTI cannot reliably be predicted with laboratory values alone, the Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score may be used by the clinician to raise suspicion of the risk of severe infection [19]. This tool should not be used to rule out NSTI. While broad-spectrum antibiotic therapy is indicated, surgical debridement is the mainstay of treatment.

KEY FACT | **Necrotizing fasciitis has a mortality rate of 30–80%, but mortality approaches nearly 100% in the absence of surgical debridement [18, 19].**

Urticaria

Pitfall | Failure to evaluate for angioedema or anaphylaxis in a patient with acute urticaria

Urticaria is a common entity, with 20% of the population experiencing hives, angioedema, or both at some point in their life [20]. Urticaria universally has wheals. Wheals are defined by three features: (1) central swelling of various sizes, with or without surrounding erythema; (2) pruritus or occasional burning sensations; and (3) the skin returning to normal appearance, usually within 1–24 hours [21]. The provider must entertain other diagnoses that can mimic urticaria such as exanthems, photosensitivity reactions, and drug hypersensitivities, particularly if individual wheals are not fleeting within 24 hours. While individual wheals are transient, acute urticaria itself can last up to six weeks.

Evaluation of the patient with acute urticaria should begin with a quick assessment of the patient's vital signs and airway status for instability, suggesting angioedema or a generalized anaphylactic reaction. Angioedema can be associated with urticaria in 50% of cases [1]. Angioedema is a distinctive reaction pattern characterized by well-circumscribed areas of edema caused by increased vascular permeability, typically involving the face and upper respiratory tract. Involvement of the upper airway can be fatal, as edema can progress to airway obstruction. The airway should be promptly secured in any patient with significant upper airway angioedema (lips, tongue, uvula, etc.). Treatment includes antihistamines, steroids, and epinephrine if there is pending airway compromise. Patients with anaphylactic reaction (urticaria, wheezing, hypotension, hypoxia) should likewise be treated with antihistamines, corticosteroids, and epinephrine and urgently transferred.

If the patient is stable, the medical history and physical examination are the best methods of determining the cause of urticaria. The most common causes of acute urticaria are outlined in Box 7.2. A careful search for these or other inciting factors should be performed.

Box 7.2 Common causes of acute urticaria

- Infections
  - Viral
  - Bacterial
  - Parasitic
- IgE-mediated allergic cases
  - Medications
  - Latex
  - Contact allergens
  - Insects (stinging, biting)
  - Foods
  - Blood products (urticarial transfusion reaction)
  - Food additives
- Direct mast cell activation
  - Certain medications (e.g. vancomycin, narcotics, succinylcholine)
  - Radiocontrast agents

Source: Adapted from Asero [22].

In most cases of urticaria, no routine laboratory tests are indicated.

Pitfall | **Failure to provide adequate antihistamine therapy in acute urticaria**

The stepwise management of acute urticaria is outlined in Box 7.3. Any identifiable underlying cause should be removed and infections should be treated, if possible. First-line therapy for urticaria is antihistamines (Table 7.1). Second-generation H_1 antihistamines are quite effective with minimal side effects. An H_2 antihistamine may be added, although data is conflicting regarding benefit [24]. If not effective, the H_2 antihistamine should be discontinued. Approximately two-thirds of cases of new-onset urticaria will be self-limited and resolve spontaneously. Initial treatment of acute urticaria should focus on the short-term relief of pruritus and angioedema, if present [22]. Patients should be instructed to start a scheduled second-generation (nonsedating) antihistamine, with ability to escalate the dose in certain adult groups. Patients at low risk of complications (e.g. young,

Box 7.3 Management of acute urticaria

- Remove identifiable underlying cause
- Treat infectious diseases
- Start a scheduled second-generation (nonsedating) antihistamine
- Instruct patient on dose escalation of nonsedating antihistamine
- Start an as needed first-generation (sedating) antihistamine
- Consider use of systemic steroids such as oral prednisone 40–60 mg daily for 5 days or equivalent (pediatrics 1 mg/kg/day × 5 days)

Table 7.1 Oral antihistamines for urticaria.

Generic name	Adult dosage	Pediatric dosage
Second-generation (nonsedating) antihistamines		
Loratadine	10 mg	2–5 yr: 5 mg daily >6 yr: 10 mg daily
Desloratadine	5 mg daily	6–11 mo: 1 mg daily 1–5 yr: 1.25 mg daily 6–11 yr: 2.5 mg daily
Cetirizine	10 mg daily	6–12 mo: 2.5 mg daily 1–5 yr: 2.5–5 mg daily 6–11 yr: 5–10 mg daily
Fexofenadine	180 mg daily	6–24 mo: 15 mg BID 2–11 yr: 30 mg BID
First-generation (sedating) antihistamines		
Diphenhydramine	25–50 mg at bedtime	2–6 yr: 6.25–12.5 mg at bedtime Max: 75 mg/day 6–12 yr: 12–25 mg at bedtime Max: 150 mg/day
Hydroxyzine	10–25 mg at bedtime	<6 yr: 12.5 mg at bedtime Max: 50 mg/day >6 yr: 12.5–25 mg at bedtime Max: 100 mg/day

BID, twice a day.
Source: Data from Dworkin *et al*. [23].

healthy patients) may find a sedating first-generation H_1 antihistamine helpful to add at bedtime.

Glucocorticoids do not appear to be necessary for isolated urticaria. However, a brief course (i.e. ≤1 week) may be added to antihistamine therapy for patients with prominent angioedema, if a patient fails scheduled antihistamines, or if symptoms persist beyond a few days.

Toxicodendron dermatitis

Pitfall | Failure to treat moderate to severe *Toxicodendron* dermatitis with adequate dose and duration of corticosteroids

The *Toxicodendron* genus of plants includes poison ivy, poison oak, and poison sumac. These plants are the most common cause of allergic contact dermatitis in North America. Up to 50 million Americans are affected each year [25]. Most adults in the United States are clinically *Toxicodendron* sensitive [25]. Urushiol is a pale yellow resin found in all parts of the *Toxicodendron* plant, including the roots, stems, leaves, and fruit. Within minutes of exposure to skin, urushiol begins to penetrate the outer skin layers and bind with cell membranes, initiating a delayed-hypersensitivity dermatitis. Symptoms of itching and erythema begin 24–48 hours after exposure (range 5 hours–15 days). This is followed by the development of vesicles, bullae, and edema. The severity and distribution of disease depend on the degree of individual sensitization, skin thickness, volume of urushiol, method of contact (direct, burning), and BSA exposed.

The diagnosis of *Toxicodendron* dermatitis is made by clinical history and examination. Most patients will recall a history of exposure in the last several days. The dermatitis caused by *Toxicodendron* plants is universally pruritic. If the patient is not complaining of itching, other diagnoses must be entertained. On examination, the eruption is asymmetric and exhibits areas of linearity where a plant or fomite rubbed the resin against the skin. Since thinner skin is more sensitive than thicker skin, patients most often have involvement on the face, neck, and dorsal extremities, with some sparing of palms and soles. In addition, it is important for the acute care provider to recognize that some patients will exhibit a phenomenon known as autoeczematization or the "id" reaction. This occurs when patients have the eruption on areas of the skin both directly exposed to *and* not directly exposed to the plants. The assessment of patients with potential *Toxicodendron* dermatitis should include vital signs and visual inspection to assess for possible secondary bacterial infection or other allergic reaction.

KEY FACT | **On examination, the eruption is asymmetric and exhibits areas of linearity where a plant or fomite rubbed the resin against the skin.**

Left untreated, *Toxicodendron* dermatitis can last three to six weeks. With treatment, symptoms may persist for two weeks. The goal of treatment is to reduce the erythema, pruritus, and blistering for the patient. Mild localized disease may be treated with potent topical corticosteroids for two to three weeks. The mainstay of therapy for widespread allergic contact dermatitis involving ≥20% of the skin surface or involvement of the face is systemic corticosteroids. For adults, a single morning dose of prednisone (1–2 mg/kg, up to a maximum dose of 60 mg) for 7–10 days, followed by a taper over the next 7–10 days, is most effective. Intramuscular steroids are also effective but there is some variability in release from the depot. Steroid dose packs do not have sufficient dose or duration and should not be used. If the patient is not treated with a high enough or long enough dose, a steroid-resistant rebound rash may occur. This is clinically similar to a *Toxicodendron* dermatitis but does not respond well to steroid treatment.

KEY FACT | **Left untreated, *Toxicodendron* dermatitis can last three to six weeks. With treatment, symptoms may persist for two weeks.**

In addition, simple steps will help ease the skin symptoms. These include (1) compresses to the skin with cool, damp white cotton towels or long underwear; (2) baths with cornstarch or colloidal oatmeal (put oatmeal in a sock and tie it to avoid clogging pipes); and (3) calamine lotion. For patients with secondary bacterial infection, use oral antibiotics and antiseptic wet dressings. Topical antihistamines, topical antibiotics,

and topical anesthetics should be avoided. Oral antihistamines decrease pruritus and allow the patient to sleep through the night.

Common infectious rashes

Tickborne illnesses

Pitfall | Failure to identify and treat a tickborne illness

Tickborne illnesses can have similar signs and symptoms, including fever/chills, myalgias, headache, fatigue, and joint pain. Distinctive rashes can be seen with Lyme disease, Southern tick-associated rash illness (STARI), RMSF, ehrlichiosis, and tularemia [26]. The most common of the tickborne diseases is Lyme disease, which results from the transmission of *Borrelia burgdorferi* to human hosts from the *Ixodes* species of tick, most commonly the deer tick [26]. Most cases are diagnosed in summer when ticks are most abundant. Early localized Lyme disease occurs 3–30 days after the tick bite and is characterized by flu-like symptoms, headaches, arthralgias, and myalgias. Approximately 80% of patients will develop the characteristic erythema migrans rash, which begins at the site of the tick bite, expanding gradually. The rash sometimes clears as it enlarges, resulting in a target or "bull's-eye" appearance, although it does not always appear as a classic erythema migrans rash. The rash may feel warm to the touch, but it is rarely pruritic or painful [26]. Early disseminated disease, occurring weeks to months after exposure, manifests with neurologic and cardiovascular pathology, including meningitis, Bell's palsy, mononeuropathies, myocarditis, and atrioventricular nodal block. Late chronic disease manifests with profound fatigue, polyneuropathy, arthralgias, encephalopathy, and leukoencephalitis [27].

The acute care provider should know the regional prevalence of Lyme and ascertain a history of potential tick exposure. If exposure is suspected, a full skin exam should be performed to check for tick(s) still attached. For patients in an endemic area presenting with symptoms of Lyme and a classic erythema migrans rash, no serologic testing is necessary. Treatment can be initiated with doxycycline 100 mg twice daily for 10 days or, alternatively, amoxicillin 500 mg three times daily for 14 days [28]. Doxycycline is the preferred agent given its efficacy against other tickborne coinfections (e.g. ehrlichiosis, anaplasmosis, etc.). Patients with symptoms of Lyme but lacking objective findings on evaluation should be referred for serologic testing. Serologic testing is wrought with complexities beyond the scope of this chapter.

If a tick has been attached for less than 48 hours, the likelihood of disease transmission is very low [6]; however, prophylaxis with a single dose of 200 mg doxycycline has been supported for nonpregnant adults and children meeting all of the following criteria: (1) the attached tick is identified as an adult or nymphal *I. scapularis* tick (deer tick); (2) the tick is estimated to have been attached for >36 hours based on degree of engorgement or time of exposure; (3) prophylaxis is begun within 72 hours of tick removal; (4) the bite occurs in a highly endemic area; and (5) doxycycline is not contraindicated [26, 29]. The Centers for Disease Control (www.cdc.gov) has the most updated guidelines around evaluation and management of tickborne diseases and providers should review the most recent recommendations.

Varicella zoster virus

Pitfall | Failure to identify high-risk patients with zoster who require immediate referral

Varicella zoster virus (VZV) is a herpes virus causing two distinct syndromes: varicella (chicken pox) and zoster (shingles). Over one million cases of shingles are diagnosed annually in the United States, with 30% of individuals developing shingles in their lifetime [30]. After primary infection with chicken pox, VZV becomes latent in the dorsal ganglia root. VZV reactivates when immunity wanes, resulting in the clinical syndrome of shingles [31]. Individuals at risk of developing shingles include those with cancer, organ transplant, human immunodeficiency virus, or those taking immunosuppressive agents [31]. Advanced age is the most significant risk factor: 40% of cases occur in people at least 60 years of age and it is estimated that approximately 50% of individuals who live to 85 years of age will have had an episode of herpes zoster in their life [31].

Clinically, shingles begins with a syndrome of rash and acute neuritis, which can precede the rash by as many as 30 days. A dermatomal erythematous

maculopapular rash is quickly followed by the development of clustered vesicles that form over a three- to five-day period and then crust over. Pain is the most common symptom of herpes zoster and it is often severe. Post-herpetic neuralgia, defined as persistent pain beyond 90 days, is one of the most debilitating complications of shingles [32].

Diagnosis of shingles can usually be made clinically by the history and the distinctive rash. Confirmatory VZV polymerase chain reaction testing can be performed and is the most sensitive technique currently available. Treatment of uncomplicated zoster is outlined in Table 7.2.

Disseminated zoster includes extensive skin involvement (more than two contiguous dermatomes, noncontiguous dermatomes, or rash on both sides of the body) and/or visceral involvement. Immunocompromised patients are high risk for disseminated disease, including those who have undergone an allogenic hematopoietic stem cell transplant within four months or with moderate to severe acute or chronic graft-versus-host disease, any transplant recipient receiving aggressive antirejection therapy, or any transplant recipient with suspected visceral dissemination (e.g. encephalitis or pneumonitis) [23]. Patients with disseminated VZV should be admitted for IV acyclovir (10 mg/kg IV every 8 hours) as most VZV-related fatalities result from disseminated infection [23].

In addition to disseminated disease, complicated zoster includes ocular manifestations, Ramsay Hunt syndrome (triad of ipsilateral facial paralysis, ear pain, and vesicles in the auditory canal and/or auricle), and neurologic complications (meningitis, encephalitis, or myelitis). Depending on the severity of these symptoms, admission for IV acyclovir and specialty consultation may be necessary.

Treating skin problems

Pitfall | **Failure to prescribe adequate and appropriate topical corticosteroids**

Many skin problems seen in the acute care setting can be adequately treated with topical corticosteroids. These include atopic dermatitis, seborrheic dermatitis, nummular eczema, psoriasis, and localized contact dermatitis. When selecting a topical corticosteroid for use, several factors must be considered: (1) What potency should be used? (2) What vehicle should be used? (3) What quantity should be dispensed?

There are several classification systems that rank topical corticosteroids by potency. In general, low-potency corticosteroids may be used on all body areas on children and on thin or occluded areas on adults (face, intertriginous, genitals). Medium-potency steroids are ideal for the trunk and extremities of teens and adults. High-potency steroids should be reserved for thick-skinned areas (palms, soles), lichenified areas, and diseases that are more corticosteroid-resistant. Because of the vast number of products available, it is reasonable for the clinician to familiarize themselves with one or two from each category for use (Table 7.3).

Second, vehicle choice is an important consideration. The most common vehicles include ointments, creams, and solutions. Ointments are water-in-oil emulsions, making them soothing, occlusive, and most effective. While greasy, they are least irritating of all vehicles and

Table 7.2 Treatment of uncomplicated zoster.

Agent	Name and dosing	Comments
Antivirals[a]	Acyclovir: 800 mg PO 5× daily for 7 d Valacyclovir: 1 g PO TID × 7 d Famciclovir: 500 mg PO TID × 7 d	All therapeutically equivalent, but valacyclovir and famciclovir preferred due to ease of dosing
Steroids[b]	Prednisone: 60 mg PO daily	Taper over 2 wk

PO, orally; TID, three times a day.

[a] Start within 72 hours of onset of rash.

[b] Avoid in patients with diabetes, hypertension, osteoporosis, and immunodeficiency (ID-1–48).

Source: Data from MICROMEDEX [33]; Tyring *et al.* [34].

Table 7.3 Topical corticosteroids.

Generic name	Vehicle: Strength
Low potency	
Desonide	Cream: 0.05% Lotion: 0.05% Ointment: 0.05%
Hydrocortisone	Cream: 0.5%, 1%, 2.5% Lotion: 1%, 2.5% Ointment: 0.5%, 1%, 2.5%
Medium potency	
Fluocinolone acetonide	Cream: 0.01%, 0.025% Ointment: 0.025% Solution: 0.01%
Triamcinolone acetonide	Cream: 0.025%, 0.1%, 0.5% Lotion: 0.025%, 0.1% Ointment: 0.025%, 0.05%, 0.1%,0.5%
High potency	
Desoximetasone	Cream: 0.05%, 0.25% Gel: 0.05% Ointment: 0.25%
Fluocinonide	Cream: 0.05% Emollient cream: 0.05% Gel: 0.05% Ointment: 0.05% Solution: 0.05%

Source: Data from MICROMEDEX [33].

will not sting when applied to inflamed skin. Creams are oil-in-water preparations that have alcohol and often propylene glycol in them. Because of this, they are not as greasy but can be irritating to cracked or fissured skin. They work well for acute and weeping dermatoses. Solutions have high amounts of alcohol, making them reasonable choices for the scalp and dense areas of hair.

Finally, it is important to consider how much BSA is involved, as this will dictate the quantity of topical corticosteroid the patient will require for therapy. Providers routinely fail to prescribe adequate amounts of topical steroids. Table 7.4 depicts the amounts required for twice-daily application for two weeks.

The most common negative side effect of short-term (two weeks) topical corticosteroid use is mild, local skin irritation. Other side effects such as skin atrophy and clinically significant hypothalamic pituitary axis suppression are unlikely with short-term use.

Table 7.4 Estimating the necessary amount of topical corticosteroid for adults.

Area	Amount for twice-daily application for 2 weeks
Face and neck	36 g
Trunk (front and back)	186 g
One arm	48 g
One hand	17 g
One leg	81 g
One foot	25 g

Source: Data from Long and Finlay [35].

Summary

The acute care provider must employ a rational approach to efficiently, safely, and effectively manage the patient who presents with a rash in the acute care setting. A series of three questions, outlined in detail in Figure 7.1, can help to define this approach. Using these questions, providers can avoid pitfalls in the management of rashes.

Pearls for improving patient outcomes

- Initiate treatment and transport immediately to a referral center for patients who present with clinical toxicity, fever, vital sign abnormalities, petechial rashes, or rashes with extensive mucosal involvement, skin sloughing, or blistering.
- Evaluate for angioedema or anaphylaxis in a patient presenting with acute urticaria.
- Instruct patients with acute urticaria to take a scheduled nonsedating antihistamine.
- Always examine carefully for mucosal membrane involvement; significant conjunctival, oral, or genital involvement should alert the provider to possible Stevens-Johnson syndrome or toxic epidermal necrolysis.
- Maintain a high index of suspicion for necrotizing soft tissue infection when pain is out of proportion.
- Identify and remove foreign bodies in patients with suspected staphylococcal toxic shock syndrome.
- Prescribe 14–20 days of systemic corticosteroids for moderate to severe *Toxicodendron* dermatitis.
- Prescribe a large enough quantity of topical corticosteroids to cover the affected body surface area for two weeks.

References

1 National Hospital Ambulatory Medical Care Survey. Emergency department summary tables. 2021. https://www.cdc.gov/nchs/data/nhamcs/web_tables/2021-nhamcs-ed-web-tables-508.pdf. Accessed March 13, 2024.

2 Centers for Disease Control and Prevention. Tickborne rickettsial diseases: Rocky Mountain spotted fever. 2024. http://www.cdc.gov/ticks/diseases/rocky_mountain_spotted_fever/. Accessed October 15, 2023.

3 Saavedra AP, Roh EK, Mikailov A. *Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology* (9th edn.). New York: McGraw Hill; 2023.

4 O'Reilly M, Paddock C, Elchos B, *et al*. Physician knowledge of the diagnosis and management of Rocky Mountain spotted fever: Mississippi, 2002. *Ann NY Acad Sci* 2003; **990**: 295–301.

5 Jay R, Armstrong PA. Clinical characteristics of Rocky Mountain spotted fever in the United States: a literature review. *J Vector Borne Dis* 2020; **57**(2): 114–120.

6 Browne BJ, Edwards B, Rogers RL. Dermatologic emergencies. *Prim Care* 2006; **33**: 685–695.

7 Heckenberg SG, de Gans J, Brouwer MC, *et al*. Clinical features, outcome, and meningococcal genotype in 258 adults with meningococcal meningitis: a prospective cohort study. *Medicine* 2008; **87**(4): 185.

8 Salzman MB, Rubin LG. Meningococcemia. *Infect Dis Clin N Am* 1996; **10**(4): 709–725.

9 Parrillo SJ. Stevens–Johnson syndrome and toxic epidermal necrolysis. *Curr Allergy Asthma Rep* 2007; **7**(4): 243–247.

10 Usatine RP, Sandy N. Dermatologic emergencies. *Am Fam Physician* 2010; **82**(7): 773–780.

11 Roujeau JC, Sterns RS. Severe adverse cutaneous reactions to drugs. *N Engl J Med* 1994; **331**(19): 1272.

12 Morales ME, Purdue GF, Verity SM, *et al*. *Am J Ophthalmol* 2010; **150**(4): 505–510.

13 Gulanikar A, Abrol A, Sagar S. Study of genital manifestations of Stevens Johnson syndrome/toxic epidermal necrolysis. *Indian J Sex Transm Dis AIDS* 2022; **43**(1): 39–42.

14 Bequignon E, Duong TA, Sbidian E, *et al*. Stevens-Johnson syndrome and toxic epidermal necrolysis: ear, nose, and throat description at acute stage and after remission. *JAMA Dermatol* 2015; **151**(3): 302–307.

15 Sekula P, Dunant A, Mockenhaupt M, *et al*. Comprehensive analysis of a cohort of patients with Stevens-Johnson syndrome and toxic epidermal necrolysis. *J Invest Demratol* 2013; **133**(5): 1197–1204.

16 Gannon T. Dermatologic emergencies. When early recognition can be lifesaving. *Postgrad Med* 1994; **96**(1): 67–70, 73–75, 79.

17 Pelletier J, Gottlieb M, Long B, *et al*. Necrotizing soft tissue infections (NSTI): pearls and pitfalls for the emergency clinician. *J Emerg Med* 2022; **62**(4): 480–491.

18 Stevens DL, Bryant AE. Necrotizing soft-tissue infections. *N Engl J Med* 2017; **377**(23): 2253.

19 Anaya DA, Dellinger EP. Necrotizing soft tissue infection: diagnosis and management. *Clin Infect Dis* 2007; **44**(5): 705.

20 Frigas E, Park MA. Acute urticaria and angioedema: diagnostic and treatment considerations. *Am J Clin Dermatol* 2009; **10**(4): 239–250.

21 Zuberbier T, Blindslev-Jensen C, Canonica W, *et al*. EAACI/GA2LEN/EDF guideline: definition, classification and diagnosis of urticaria. *Allergy* 2006; **61**(3): 316–320.

22 Asero R. New-onset urticaria. 2022. https://pro.uptodatefree.ir/show/8101. Accessed October 15, 2023.

23 Dworkin RH, Johnson RW, Breuer J, *et al*. Recommendations for the management of herpes zoster. *Clin Infect Dis* 2007; **44**(Suppl 1): S1.

24 Fedorowicz Z, van Zuuren EJ, Hu N. Histamine H2-receptor antagonists for urticaria. *Cochrane Database Syst Rev* 2012; **2012**(3): CD008596.

25 Gladman AC. Toxicodendron dermatitis: poison ivy, oak, and sumac. *Wilderness Environ Med* 2006; **17**(2): 120–128.

26 Centers for Disease Control and Prevention. Ticks home. n.d. https://www.cdc.gov/ticks/index.html. Accessed March 20, 2024.

27 Bratton RL, Corey R. Tick-borne disease. *Am Fam Physician* 2005; **71**(12): 2323–2330.

28 Hu L, Shapiro ED. Treatment of Lyme disease. 2018. https://uptodatefree.ir/topic.htm?path=treatment-of-lyme-disease. Accessed October 15, 2023.

29 Lantos PM, Rumbaugh J, Bockenstedt LK, *et al*. Clinical practice guidelines by the Infectious Diseases Society of America (IDSA), American Academy of Neurology (AAN), and American College of Rheumatology (ACR): 2020 guidelines for the prevention, diagnosis, and treatment of Lyme disease. *Arthritis Rheumatol* 2021; **73**(1): 12–20.

30 Harpaz R, Ortega-Sanchez IR, Seward JF. Prevention of herpes zoster: recommendations of the Advisory Committee of Immunization Practices (ACIP). *MMWR* 2008; **57**(RR-5): 1–30.

31 Schmader K. Herpes zoster in older adults. *Clin Infect Dis* 2001; **32**(10): 1481–1486.

32 Forbes HJ, Bhaskaran K, Thomas SL, *et al*. Quantification of risk factors for postherpetic neuralgia in herpes zoster patients: a cohort study. *Neurology* 2016; **87**(1): 94–102.

33 MICROMEDEX. n.d. www.micromedexsolutions.com. Accessed October 15, 2023).

34 Tyring SK, Beutner KR, Tucker BA, *et al*. Antiviral therapy for herpes zoster: randomized, controlled clinical trial of valacyclovir and famciclovir therapy in immunocompetent patients 50 years and older. *Arch Fam Med* 2000; **9**(9): 863–869.

35 Long CC, Finlay AY. The finger-tip unit – a new practical measure. *Clin Exp Dermatol* 1991; **16**(6): 444–447.

CHAPTER 8
Management of Common Infections

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Introduction

The management of common infectious diseases is central to the practice of urgent care medicine. Acute care providers are challenged to identify the varying manifestations of some of these infectious diseases and treat them accordingly. In all cases, providers are faced with the task of choosing appropriate antibiotics when indicated, while withholding antibiotics in instances where they are not needed. The goal of this chapter is to review some of the most common infectious diagnoses in urgent care settings with a focus on their accurate diagnosis and appropriate treatments.

Pitfall | **Inappropriate use of incision and drainage or antibiotics to treat cutaneous abscesses**

Epidemiologic data demonstrates that more than 50% of skin and soft tissue infections (SSTIs) at major medical centers in the United States are a result of community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) [1]. Patients can present with furuncles, folliculitis, impetigo, purulent cellulitis, carbuncles, paronychia, abscesses, and necrotizing fasciitis. The role of CA-MRSA in nonpurulent cellulitis is less clear [2]. The key points of the Infectious Diseases Society of America (IDSA) guidelines for evidence-based management of SSTIs are summarized in Table 8.1.

Incision and drainage (I&D) is the treatment of choice for most simple skin abscesses [4]. However, I&D is invasive and is the second most painful procedure performed in the emergency department (ED) [5]. There are potential alternatives to I&D for abscess management in some select cases [6, 7]. One alternative to I&D is treatment with antibiotics alone in very small abscesses (<1.3 cm in diameter and <0.4 cm in depth) in otherwise healthy patients who are averse to I&D. However, there is an 8% treatment failure with this approach, so appropriate follow-up is important [7].

Loop drainage has emerged as a promising alternative to I&D for larger (>5 cm) abscesses. Loop drainage is less invasive than I&D because it involves making two small incisions at each end of the abscess rather than one larger incision. A "loop" is fed through the two incisions and tied to keep the abscess cavity open and facilitate drainage (see Figure 8.1). Either the cuff of a glove or a vessel loop can be used as the loop in this procedure [8, 9]. Several studies have found that loop drainage is an effective or superior treatment compared to I&D [10–13]. A 2020 randomized controlled trial in adults demonstrated that loop drainage is noninferior to I&D in abscess resolution. Interestingly, participants in the loop drainage treatment group had significantly fewer complications and return visits than participants in the I&D treatment group. Of note is that the authors report the inclusion of many small abscesses (36% in the loop drainage group <3 cm, 44.7% in the I&D group <3 cm) [14]. In summary, loop drainage is a promising technique and further research should investigate its effectiveness for very large (>10 cm) abscesses.

Complicated SSTIs are characterized by more severe disease, systemic symptoms, and progressive examination findings. Patient characteristics that predispose to complicated SSTIs include a history of immunosuppression, diabetes mellitus, peripheral vascular disease

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Table 8.1 Recommended initial treatment of skin and soft tissue infections (SSTIs) based on patient and infection characteristics.

Recommended initial treatment	SSTI and patient characteristics
I&D only	Simple abscess in otherwise healthy patient
I&D + antibiotics for CA-MRSA	Abscess with one or more of the following: • Severe or extensive disease • Progressive associated cellulitis • Signs of systemic illness • High-risk comorbidities or immunosuppression • Extremes of age • Problematic location for I&D (hands, face, genitals) • Associated septic phlebitis • Ineffective previous I&D
Empiric antibiotics for CA-MRSA without I&D	Cellulitis with one or more of the following: • Purulence but no abscess amenable to drainage • Purulence with small abscess and preference not to perform I&D • No purulence but no response to beta-lactam treatment • Signs of systemic illness
Empiric antibiotics for beta-hemolytic strep	Nonpurulent cellulitis

CA-MRSA, community-acquired methicillin-resistant *Staphylococcus aureus*; I&D, incision and drainage.
Source: Data from Liu *et al*. [3].

involving the affected area, or extremes of age. Infection-specific characteristics signaling a more complicated case include abscess size >5 cm, extensive surrounding cellulitis, location in an area not amenable to I&D, and the presence of systemic signs of infection such as fever and tachycardia. Antimicrobial therapy is indicated in these cases (see Table 8.1) [3] and appropriate antibiotic choice is of the utmost importance. SSTIs presenting as classic CA-MRSA infections, such as simple abscesses, furuncles, or carbuncles, should be treated with an agent that covers CA-MRSA with appropriate dosing (see Tables 8.2 and 8.3 for specific antibiotics and dosing). Of note is that linezolid has numerous drug interactions, therefore it is recommended only after treatment failure with other agents and it should only be prescribed after close review of all current medications for interactions.

KEY FACT | **Selection of antibiotics for skin and soft tissue infections varies based on key infection characteristics.**

SSTIs that could also involve beta-hemolytic streptococcal infection, most notably purulent cellulitis or extensive cellulitis surrounding an abscessed or necrotizing area, require appropriate coverage for both CA-MRSA and strep, as indicated in Table 8.2. Nonpurulent cellulitis alone is not commonly associated with CA-MRSA, therefore antibiotics covering strep alone are recommended. Contrary to common understanding, trimethoprim-sulfamethoxazole (TMP-SMX), like clindamycin, is able to provide adequate strep and CA-MRSA coverage as a single agent [15]. Doxycycline and minocycline require the addition of a beta-lactam (e.g. amoxicillin or cephalexin) to the regimen for streptococcal coverage [3]. Efforts aimed at preventing the recurrence of CA-MRSA infections have focused on patient education. The important points are summarized in Box 8.1.

Antibiotics have not generally been considered a requirement for the treatment of uncomplicated skin abscesses. Many randomized controlled trials have previously indicated that I&D alone is sufficient for the treatment of uncomplicated abscesses, and that antibiotics do not improve cure rates [1, 16, 17]. In line with these findings, a 2013 survey distributed to 15 EDs across the United States showed that only 17% of providers routinely used antibiotics in the management of an uncomplicated abscess [18]. Furthermore, IDSA guidelines state that antibiotics should be reserved for complicated cases, as noted earlier [4].

In 2016, Talan *et al*. conducted a randomized controlled trial comparing TMP-SMX to placebo for the management

Figure 8.1 Steps in loop drain for treatment of large abscesses. *Source*: Photo by Siamak Moayedi MD (Chapter author).

of "uncomplicated" abscesses [19]. Importantly, this study included abscesses of all sizes and febrile patients. The rate of clinical cure was higher in the TMP-SMX group compared with the placebo group (80.5% versus 73.6%). There were slightly more gastrointestinal symptoms in the TMP-SMX group [19]. A subgroup analysis revealed that the TMP-SMX treatment effect was largest in cases where participants had a fever, positive MRSA culture, or history of MRSA infection [20].

In another large, multicenter trial to examine the utility of antibiotics after I&D, the cure rate was higher in participants who received clindamycin (83.1%) or TMP-SMX (81.7%) compared with placebo (68.9%) [21]. This study excluded complicated cases (abscess >5 cm in diameter, immunocompromised patients, those with diabetes, and patients with systemic symptoms). Of note is that the beneficial effect of clindamycin should be

Table 8.2 Infectious Diseases Society of America oral antibiotic recommendations for coverage of skin and soft tissue infections in outpatients.

Likely organism	Agents recommended
CA-MRSA	Trimethoprim-sulfamethoxazole (TMP-SMX) Clindamycin Doxycycline or minocycline Linezolid
CA-MRSA + beta-hemolytic strep	TMP-SMX Clindamycin Doxycycline or minocycline + beta-lactam[a] Linezolid

CA-MRSA, community-acquired methicillin-resistant *Staphylococcus aureus*.
[a] Beta-lactam agents include penicillins, cephalosporins and carbapenems.
Source: Data from Liu *et al*. [3] and McCreary *et al*. [15].

Table 8.3 Antimicrobial agents and dosing for outpatient management of CA-MRSA skin and soft tissue infections.

Antimicrobial	Dosing
Trimethoprim-sulfamethoxazole (TMP-SMX)	1 tablet PO BID
Clindamycin	150 mg–450 mg PO q 6 h
Doxycycline	100 mg PO BID
Minocycline	100 mg PO BID (first dose 200 mg)
Linezolid	400 mg PO q 12 h

BID, twice a day; CA-MRSA, community-acquired methicillin-resistant *Staphylococcus aureus*; PO, orally; q, every.
Source: Data from Liu *et al*. [3].

weighed against the increased risk of adverse events. Approximately 1 in 5 patients in the clindamycin treatment group experienced an adverse event, most commonly gastrointestinal symptoms [21].

While these studies provide compelling evidence for the benefit of antibiotics in abscess management, there is still debate regarding universal antibiotic use after I&D. In 2018, Pulia *et al*. found that additional antibiotic prescriptions, repeat drainage procedures, and return visits to the ED did not differ significantly between patients who had been prescribed antibiotics in their

Box 8.1 Areas for focus of patient education to prevent recurrent community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections

Keep draining wounds covered with clean, dry bandages.

Maintain good personal hygiene including regular bathing and hand washing with soap and water or an alcohol-based hand gel, especially after contact with wound.

Avoid reusing or sharing personal items (e.g. razors, towels) that have contacted infected skin.

Focus cleaning efforts on high-touch areas that may contact bare skin or uncovered infections (e.g. door knobs, phones, counter tops, toilet seats).

Source: Data from Liu *et al*. [3].

initial management and those who received drainage alone [22]. None of these studies detected a difference in hospitalizations between patients who received antibiotics and those who did not [19, 22]. Pulia and Fox advocate against the routine use of antibiotics in abscess management because the risks may outweigh the benefits [23].

KEY FACT | **The benefits of antibiotic prescription after incision and drainage are debatable and likely only apply to complicated abscesses.**

In summary, there is some evidence to support the use of antibiotics after an I&D procedure, especially in complicated cases. Expanding the use of antibiotics in a more universal manner contributes to the population-level risk of antibiotic resistance, thereby diminishing the effectiveness of available antibiotics. Antibiotics also confer risks at the individual level, such as the risk of *Clostridioides difficile* (formerly known as *Clostridium difficile*) infection [23]. Furthermore, Conway and Friedman point out that the rate of clinical cure for skin abscess without antibiotic treatment is quite high (84%), and the consequences of clinical failure may not be serious enough to warrant the routine use of antibiotics in abscess management [24].

Pitfall | Misdiagnosis of an aggressive soft tissue infection as cellulitis

Uncomplicated cellulitis in a patient without immunosuppression is commonly treated with outpatient antibiotics. It is imperative to always consider more aggressive soft tissue infections, as delay in diagnosis and management of necrotizing soft tissue infections (NSTIs) will lead to significant morbidity and mortality. NSTIs include necrotizing fasciitis, myositis, and cellulitis. NSTIs lead to rapidly progressive tissue destruction and have a mortality of more than 20% [25]. The difficulty in diagnosis of this entity is related to the pathogens' ability to spread along tissue planes rapidly and widely without significant involvement of the skin surface. The most common pathogens leading to NSTIs are beta-hemolytic streptococci, *Bacteroides, Escherichia coli*, *Clostridium*, and various mixtures of anaerobic organisms. The subtypes of these bacteria capable of producing necrotizing fasciitis possess exotoxins that cause tissue destruction.

KEY FACT | **Necrotizing fasciitis rapidly spreads along tissue planes without significant skin surface involvement.**

Distinguishing an early NSTI that would require emergent surgical debridement and aggressive parenteral antibiotics from an uncomplicated cellulitis is challenging. Clinical signs that are specific for NSTI include the presence of bullae, ecchymosis, crepitus, and superficial anesthesia of the skin. Unfortunately, these signs occur in a minority of cases and develop late in the process of the disease. Clinical clues that can help to differentiate between an NSTI and cellulitis early in the process include pain out of proportion to the extent of skin involvement, edema extending past the skin erythema, and a history of immunosuppression (specifically poorly controlled diabetes). However, it is important to recognize that NSTIs can occur among healthy individuals with no past medical history or portal of entry [25]. Patients with signs concerning for a NSTI should be referred immediately to an ED to initiate emergent care and diagnostic measures.

Pitfall | Misdiagnosis of deep venous thrombosis as cellulitis

Cellulitis in the lower leg is characterized by signs and symptoms that may be similar to those of a deep vein thrombosis (DVT), such as warmth, pain, and swelling. Given the significant mortality associated with untreated DVT, it is important to make an accurate diagnosis. Although fever and chills are more common with cellulitis, these findings can occur with DVT. Lymphangitis

and tender adenopathy in the ipsilateral groin support the diagnosis of cellulitis. Furthermore, cellulitis will have sharply demarcated and advancing erythematous borders.

In one study of ED patients presenting with lower-extremity pain and swelling with an initial impression of DVT versus cellulitis, 66% were diagnosed with cellulitis, 17.5% had a confirmed DVT, 2% were diagnosed with both cellulitis and DVT, and 16.5% were diagnosed with another entity such as Baker's cyst or superficial thrombosis. Patients with a diagnosis of cellulitis were more likely to have a history of diabetes. Interestingly, patients with leukocytosis were less likely to have cellulitis. This affirms the futility of ordering complete blood counts to differentiate between cellulitis and DVT [26]. Studies documenting the prevalence of both cellulitis and acute DVT in the same lower extremity range from 0 to 15% [27]. It is plausible that the edema caused by an occlusive DVT can place patients at risk for developing cellulitis. In this scenario, a careful history will likely illustrate the presence of edema prior to the secondary infection.

KEY FACT | **The prevalence of both cellulitis and acute deep vein thrombosis in the same extremity is 0–15%.**

Pitfall | **Overuse of antibiotics for upper respiratory infections**

Commonly, antibiotics prescribed in acute care practice are for upper respiratory infections (colds), sinusitis, pharyngitis, and bronchitis. For most of these conditions, antibiotics do not provide any benefit, while contributing to antibiotic resistance and placing the patient at risk of medication side effects. Furthermore, the prescription rates for expensive and broad-spectrum antibiotics have been increasing, creating an undue financial burden on patients and healthcare systems.

There is ample evidence-based literature guiding the appropriate management of these common maladies (listed in the following sections). However, the trend for antibiotic misuse continues due to a variety of pressures, including patients who insist on antibiotics (reinforced from previous prescriptions), lack of time to explain why antibiotics are unnecessary, commercial advertising from drug companies and providers who are overly cautious [28]. There are a variety of ways to decrease inappropriate antibiotic prescription. Using several methods in combination works better than using one method alone [28]. Notably, patient satisfaction with their care for these conditions is predominantly based on provider–patient communication, rather than antibiotic prescription.

KEY FACT | **Patient satisfaction is predominantly based on provider–patient communication rather than antibiotic prescription.**

Five communication strategies are listed in Box 8.2. It is often helpful to personalize the risk of antibiotic use by highlighting common side effects such as gastrointestinal upset, yeast infection, or the development of resistant strains of pathogens risking treatment failures for future serious bacterial infections. Other strategies include giving patients printed material supporting your decision not to prescribe antibiotics. Finally, another effective method of decreasing antibiotic use is asking a patient to wait for a set number of days and only to fill their prescription if their symptoms do not improve after that time.

Box 8.2 Communication strategies to decrease antibiotic prescription

- *Provide a specific diagnosis* to help patients feel validated. For example, say "viral bronchitis" instead of referring to an illness as "just a virus."
- *Recommend symptomatic relief*. Often patients request an antibiotic because they think it will help them feel better, without realizing that there are effective symptomatic therapies.
- *Share normal findings* as you go through your exam. For example, let patients know that their lungs sound clear. This reassures the patient that the illness may not be as severe as they thought and may make them more open to the idea that they do not need an antibiotic.
- *Discuss potential side effects* of antibiotic use, including cost, adverse events (e.g. rash, vomiting, diarrhea, yeast vaginitis) and resistance.
- *Explain to the patient what to expect* over the next few days. This can help them feel reassured and empowered. Give patients a plan of action in case symptoms do change or become worse.

Source: Data from Centers for Disease Control and Prevention, http://www.cdc.gov/antibiotic-use

Antibiotic therapy

Principles for appropriate antibiotic therapy for evaluation and treatment of bronchitis, pharyngitis, sinusitis, and upper respiratory infections are summarized in the following sections. These guidelines apply to otherwise healthy adults and exclude the elderly and patients with comorbid conditions such as immunosuppression or emphysema.

Bronchitis

The evaluation of adults with an acute cough illness should focus on ruling out pneumonia. In the healthy, nonelderly adult, pneumonia is uncommon in the absence of vital sign abnormalities or asymmetric lung sounds, therefore chest radiography is usually not indicated. The presence of additional viral symptoms such as nasal/sinus congestion and sore throat makes viral bronchitis much more likely. Colored sputum does not indicate bacterial infection. Cough duration does not dictate the need for chest radiography. Antibiotic treatment of uncomplicated bronchitis is not recommended, regardless of the duration of the cough [29]. Cough suppressants, first-generation antihistamines, and decongestants provide symptomatic relief in some cases [30].

Pharyngitis

Group A beta-hemolytic *Streptococcus* (GABHS) is the etiologic agent in approximately 10% of adult cases of pharyngitis. Most adults with acute pharyngitis (including GABHS) have a self-limiting illness. The benefits of antibiotic treatment of adult pharyngitis apply only to those patients with GABHS infection. All patients with pharyngitis should be offered appropriate doses of analgesics, antipyretics, and other supportive care. Clinically screen all adult patients with pharyngitis for the presence of the four Centor criteria [31]: (1) fever or recent history of fever; (2) tonsillar exudates; (3) absence of cough; and (4) tender anterior cervical lymphadenopathy. Test patients for GABHS with two or three criteria using a rapid antigen test. Limit antibiotic therapy to patients with a positive test or patients with four criteria. If a rapid antigen test is unavailable, limit antibiotic therapy to patients with three or four criteria. The preferred antibiotics for treatment of acute GABHS pharyngitis are penicillin or amoxicillin for 10 days [32]. In penicillin-allergic patients, cephalexin or clindamycin is preferable given that resistance to azithromycin and clarithromycin is increasing [30]. Corticosteroids are not indicated in the routine treatment of exudative pharyngitis and only provide a small reduction in the time to pain relief in GABHS pharyngitis [33].

KEY FACT | **Corticosteroids are only beneficial for exudative pharyngitis caused by group A beta-hemolytic Streptococcus.**

Sinusitis

Over 90% of cases of acute sinusitis are due to uncomplicated viral infections. The clinical diagnosis of acute bacterial sinusitis should be reserved for patients with sinusitis symptoms that are severe, including fever >39 °C (102 °F) and purulent nasal discharge or facial pain lasting four days or more. Other diagnostic signs of bacterial sinusitis include persistent symptoms for more than ten days without improvement or clinical worsening after initial improvement of symptoms. Antibiotics are not guaranteed to help even if a bacterial cause is suspected. Watchful waiting is an effective strategy for uncomplicated cases with reliable follow-up. Initial antibiotic treatment should be with the most narrow-spectrum agent that is active against the likely pathogens, *Streptococcus pneumoniae* and *Haemophilus influenza* [30, 34]. Initial choices include standard amoxicillin or amoxicillin-clavulanate in patients with risk factors for pneumococcal resistance (i.e. local resistance rates >10%, age ≥65 years, hospitalization in the last five days, antibiotic use in the previous month, immunocompromising comorbidities, severe infection, evidence of systemic toxicity with temperature of ≥102 °F) [34]. Penicillin-allergic patients can take doxycycline or a respiratory fluoroquinolone. Azithromycin is not recommended due to high rates of antibiotic resistance [30].

Upper respiratory tract infections

An upper respiratory tract infection is defined as an acute infection that is typically viral in origin and in which sinus, pharyngeal, and lower airway symptoms, although frequently present, are not prominent. At least 200 different viruses can cause these symptoms [30]. Antibiotic treatment of nonspecific upper respiratory infections in adults does not expedite illness resolution or prevent complications and is therefore not recommended. Purulent secretions in the nares and throat or changes in sputum color from yellow to green neither

predict bacterial infection nor benefit from antibiotic treatment [35]. Decongestants combined with an antihistamine may provide short-term symptom relief [36].

KEY FACT | **Change in color of sputum from yellow to green does not predict the presence of a bacterial infection and does not suggest any benefit from antibiotics.**

Pitfall | Failure to diagnose and appropriately treat *Clostridioides difficile* in patients presenting with diarrhea

An increasing number of patients ultimately diagnosed with *C. difficile* infection present without a history of traditional risk factors, such as recent use of antibiotics or hospitalization. Other sources of *C. difficile* thought to possibly be responsible for the emergence of this new strain of community-acquired *C. difficile* (CACD) can include soil, household pets, food, or secondhand exposure to other colonized individuals [37, 38].

While the incidence of healthcare-associated *C. difficile* declined between 2011 and 2017, there was no significant change in the incidence of community-acquired infection [39]. Some studies have shown that up to 36% of patients with CACD had not received any antibiotics in the preceding 12 weeks prior to diagnosis and 18% had no recent healthcare exposure [40]. Overall, when patient characteristics were analyzed, it appears that patients presenting with diarrhea from CACD are younger than the traditional *C. difficile* population and have less severe clinical disease [38]. While patients with a history of prior hospitalizations and antibiotic use (especially clindamycin or a fluoroquinolone) remain at increased risk of *C. difficile*, other risk factors such as the use of proton pump inhibitors, a history of concurrent renal failure, and a history of irritable bowel disease have been identified in patients with CACD [37, 38].

KEY FACT | **36% of patients with community-acquired C. difficile have not been exposed to antibiotics in the 12 weeks prior to symptom development and 18% have no recent healthcare exposure.**

The changing epidemiology of patients diagnosed with CACD raises the importance of considering and testing for *C. difficile* in the urgent care setting. Any patient suffering from more than three watery stools per day for at least two days may be diagnosed with *C. difficile* and testing should be considered [37].

When initiating outpatient therapy to treat the first episode of uncomplicated *C. difficile*–associated diarrhea, providers may prescribe oral vancomycin or oral fidaxomicin. Uncomplicated episodes are characterized by a white blood cell count less than 15,000 cells/ml and serum creatinine less than 1.5 mg/dl. While there is a preference for oral fidaxomicin per the 2021 IDSA guidelines to decrease the risk of recurrence, lack of pharmacy availability of this agent may limit its use [41]. Oral vancomycin is an acceptable alternative for a first infection if fidaxomicin is unavailable. Oral metronidazole is only recommended as a treatment option in settings where vancomycin and fidaxomicin are unobtainable. If prescribed, oral metronidazole should be taken for 10–14 days. Details of dosing and duration of treatment for an initial, uncomplicated episode of *C. difficile* are provided in Table 8.4 [41]. Severe, recurrent, or complicated infections should be referred to the ED for evaluation and treatment. There is no evidence to support the use of probiotics such as *Lactobacillus* in *C. difficile* treatment [37, 42].

Pitfall | Overreliance on urine dipstick results in treating women for simple urinary tract infections

Dysuria and urinary frequency are common urgent care complaints in female patients. The decision regarding treatment of these symptoms with antibiotics can be difficult, as acute care providers must weigh the risks of inappropriate antibiotic treatment against patient expectations. A urine dipstick can be helpful in diagnosing urinary tract infections (UTIs) in women with positive

Table 8.4 Antimicrobial agents and dosing for outpatient management of uncomplicated, initial *C. difficile* infection.

Antimicrobial	Dosing
Fidaxomicin	200 mg PO BID × 10 d
Vancomycin	125 mg PO QID × 10 d
Metronidazole (only if the other agents are not available)	500 mg PO TID × 10–14 d

BID, twice a day; PO, orally; QID, four times a day; TID, three times a day.

nitrates or leukocytes, but there are many patients with typical symptoms and nondiagnostic dipstick results [43].

KEY FACT | **Empiric antibiotics for dysuria and urinary frequency can decrease the duration of symptoms irrespective of urine dipstick results.**

One prospective double-blinded randomized placebo-controlled clinical trial assessed the value of antibiotics for women between 16 and 50 presenting with symptoms of dysuria and frequency but negative urine dipstick results. Women in this trial were assigned to receive three days of trimethoprim or placebo. The women treated with trimethoprim had two fewer days of dysuria and four fewer days of constitutional symptoms such as fever and chills [44].

These results demonstrate that symptoms of dysuria, urinary frequency, and urinary urgency, in the absence of vaginal discharge and risk of sexually transmitted infections, warrant antibiotic treatment for uncomplicated UTI irrespective of the urine dipstick results [43]. Clearly, this information should be weighed against the risks of adverse medication reactions, superinfection, and bacterial resistance to antibiotics in a given community [44].

Pitfall | Confusion of scabies infestation with bed bug infestation

Bed bugs (*Cimex lectularius*) are wingless, red-brown, blood-sucking insects that grow up to 7 mm in length with a lifespan of between four months and one year. They typically hide during daylight and emerge nocturnally to feed. They are capable of anesthetizing the skin, which allows them to take a blood meal without detection. Bed bug bites are occasionally observed as linear (groups of three), erythematous, and pruritic lesions ranging from macules to wheals resembling mosquito bites [45]. Symptomatic therapy with antihistamines and topical steroids is beneficial. Eradication requires elimination of the bugs from bedding and other hiding places. Powerful insecticides and professional exterminators are often required for this task.

Scabies is caused by microscopic mites too small to be seen without magnification. They burrow into the superficial layer of the dermis and lay eggs. Scabies is spread by direct and prolonged skin-to-skin contact. It occurs most commonly in institutional environments such as homeless shelters, schools, and nursing homes. Patients exposed for the first time do not develop any symptoms for two to six weeks after infestation. Infested patients can transmit the mites during this asymptomatic period. Patients with prior exposure to mite antigens will develop pruritis and skin findings between one and four days post infestation.

The intense itching and skin rash are caused by a delayed allergic reaction to the feces and proteins of the mites. Itching is intensified at nighttime and during physical activity when core temperature is elevated. The rash appears as nonspecific small papules that may be diffuse or limited to areas of the body where two layers of skin rub or touch each other (e.g. between fingers, armpits, buttocks, inguinal area, etc.). Occasionally tiny burrows can be visualized as raised lines on the skin surface. However, burrows may be difficult to find. The most common complication is superinfection of the skin caused by traumatic excoriation. The diagnosis of scabies is clinical as skin evaluation for mites has a low yield.

Treatment is recommended for all patient contacts with prolonged skin-to-skin exposures (recall that patients infested for the first time may be asymptomatic for up to six weeks). The rash and pruritis may persist for two weeks post treatment. First-line therapy is topical 5% permethrin cream applied to the body for eight hours. Lindane solution is not suggested as a first-line therapy due to its potential for neurotoxicity. The absorption of lindane is enhanced in cases of severe excoriation. Lindane is contraindicated during pregnancy and in children under five years of age. Refractory and severe cases can be simultaneously treated with oral ivermectin [45]. Bedding and clothing used by the patient can be placed in a dryer and exposed to high heat or bagged for three days, as mites are unable to survive without skin exposure for that length of time.

KEY FACT | **Scabies mites are unable to survive without skin exposure for more than three days.**

The difference between bed bug bites and scabies can be subtle. In general, bed bug bites often occur in a linear pattern. Furthermore, bed bugs do not burrow under the dermis and thus are not contagious unless they are hidden in clothing or bedding. Scabies mites are not visible to the naked eye, whereas bed bugs can be seen. The pruritis associated with bed bug bites is

improved with warm water, whereas it is intensified if related to scabies.

Pitfall | **Indiscriminate use of antibiotics as prophylaxis after tick bites**

Lyme disease, caused by the spirochete *Borrelia burgderferi*, is transmitted by the *Ixodes* tick, commonly known as the deer tick. Most cases of Lyme disease in the United States occur in the northeast, from Maryland to New England, as well as parts of Minnesota, Wisconsin, and Michigan [46]. Due to aggressive Lyme disease information campaigns, patients often present to acute care facilities with concerns regarding tick exposure, ranging from the observation of a tick on the skin to the complaint that a tick is embedded in their skin. Routine use of antibiotics to prevent Lyme disease is not recommended because of the overall low risk of transmission (1–3%) [46].

KEY FACT | **Transmission of Lyme disease requires tick attachment and engorgement for more than 36 hours.**

Merely seeing a tick or even having one attached for less than 36 hours is not an indication for treatment. The spirochetes need time to replicate and multiply in the gut of the tick before they can move to its salivary gland to be regurgitated into the patient's bloodstream. This process takes more than 36–48 hours and necessitates engorgement of the tick with blood.

Prophylaxis is recommended if the criteria in Box 8.3 are met. If the patient does meet the criteria for prophylaxis, a single 200 mg dose of doxycycline is effective in preventing Lyme disease in adults (4.4 mg/kg for children of any age weighing <45 kg) [47, 48]. There is no indication for serum testing as antibodies to *B. burgderferi* antigens do not form for several weeks. Ticks that are embedded in the skin must be removed, ensuring that there is no compression of the gut. Techniques such as squeezing or burning are discouraged as they may increase the risk of transmission. The correct method for removing a tick involves the use of fine tweezers, grasping the tick as closely as possible to the head and pulling straight out without twisting. If a piece of the head is retained, there is no indication for aggressive measures such as coring the skin. Simply leave the head in place and it will eventually be pushed out by the skin.

Box 8.3 Criteria for Lyme prophylaxis of tick bites

- The tick is identified to be an *Ixodes* species.
- The exposure was in a Lyme-endemic region.
- The tick's body was engorged with blood (not flat).
- The tick was removed within the past 72 hours.
- The patient has no contraindications to doxycycline therapy (allergy to doxycycline, pregnant or lactating women).

Regardless of antibiotic prophylaxis, patients should be instructed to look for the classic erythema migrans rash that occurs in 80% of Lyme infections. The rash can occur any time within the first month of exposure and once present rapidly expands into the typical target appearance. It is possible for the rash to be uniformly erythematous and may be pruritic or painful. The presence of this rash necessitates Lyme disease treatment (doxycycline for 10–14 days, amoxicillin for 14 days, or cefuroxime for 14 days) [48]. Doxycycline is considered safe regardless of age for up to 21 days [15].

Pearls for improving patient outcomes

- Antibiotics are not indicated after incision and drainage for treatment of uncomplicated abscesses.
- Consider necrotizing fasciitis when there is pain out of proportion to the extent of skin involvement or edema extends past the cellulitis margins.
- Do not rely on a white blood cell count to differentiate between deep vein thrombosis and cellulitis.
- Personalize the risk of antibiotic use by highlighting common side effects and the development of resistance leading to treatment failures in the future.
- Do not prescribe corticosteroids for symptom relief in exudative pharyngitis caused by viruses.
- Patients without a history of recent antibiotic use or hospitalization are still at risk of diarrhea from community-acquired *Clostridioides difficile*.
- Women with urinary symptoms but without vaginal symptoms will likely benefit from a short course of antibiotics for presumed urinary tract infection, even in the absence of a negative urine dipstick.
- Consider the diagnosis of bed bugs over scabies if the patient reports seeing bugs and the pruritis improves with warm water.
- Deer tick exposure/attachment for less than 36 hours does not require Lyme disease prophylaxis.

References

1 Schmitz GR, Bruner D, Pitotti R, *et al*. Randomized controlled trial of trimethoprim-sulfamethoxazole for uncomplicated skin abscesses in patients at risk for community-associated methicillin-resistant *Staphylococcus aureus* infection. *Ann Emerg Med* 2010; **56**(3): 283–287.

2 Wallin TR, Hern HG, Frazee BW. Community-associated methicillin-resistant *Staphylococcus aureus*. *Emerg Med Clin North Am* 2008; **26**(2): 431–455. ix.

3 Liu C, Bayer A, Cosgrove SE, *et al*. Clinical practice guidelines by the Infectious Diseases Society of America for the treatment of methicillin-resistant *Staphylococcus aureus* infections in adults and children: executive summary. *Clin Infect Dis* 2011; **52**: 285–292.

4 Stevens DL, Bisno AL, Chambers HF, *et al*. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis* 2014; **59**: e10–e52.

5 Singer AJ, Richman PB, Kowalska A, *et al*. Comparison of patient and practitioner assessments of pain from commonly performed emergency department procedures. *Ann Emerg Med* 1999; **33**: 652–658.

6 Menegas S, Moayedi S, Torres M. Abscess management: an evidence-based review for emergency medicine clinicians. *J Emerg Med* 2021; **60**(3): 310–320.

7 Russell FM, Rutz M, Rood LK, *et al*. Abscess size and depth on ultrasound and association with treatment failure without drainage. *West J Emerg Med* 2020; **21**: 336–342.

8 Tsoraides SS, Pearl RH, Stanfill AB, *et al*. Incision and loop drainage: a minimally invasive technique for subcutaneous abscess management in children. *J Pediatr Surg* 2010; **45**: 606–609.

9 Thompson DO. Loop drainage of cutaneous abscesses using a modified sterile glove: a promising technique. *J Emerg Med* 2014; **47**: 188–191.

10 McNamara WF, Hartin CW, Escobar MA, *et al*. An alternative to open incision and drainage for community-acquired soft tissue abscesses in children. *J Pediatr Surg* 2011; **46**: 502–506.

11 Ladde JG, Baker S, Rodgers CN, *et al*. The LOOP technique: a novel incision and drainage technique in the treatment of skin abscesses in a pediatric ED. *Am J Emerg Med* 2015; **33**: 271–276.

12 Ozturan IU, Dogan NO, Karakayalı O, *et al*. Comparison of loop and primary incision & drainage techniques in adult patients with cutaneous abscess: a preliminary, randomized clinical trial. *Am J Emerg Med* 2017; **35**: 830–834.

13 Gottlieb M, Peksa GD. Comparison of the loop technique with incision and drainage for soft tissue abscesses: a systematic review and meta-analysis. *Am J Emerg Med* 2018; **36**: 128–133.

14 Schechter-Perkins EM, Dwyer KH, Amin A, *et al*. Loop drainage is noninferior to traditional incision and drainage of cutaneous abscesses in the emergency department. *Acad Emerg Med* 2020; **27**(11): 1150–1157.

15 McCreary EK, Johnson MD, Jones TM, *et al*. Antibiotic myths for the infectious disease clinician. *Clin Infect Dis* 2023; **77**(8): 1120–1125.

16 Rajendran PM, Young D, Maurer T, *et al*. Randomized, double blind, placebo-controlled trial of cephalexin for treatment of uncomplicated skin abscesses in a population at risk for community acquired methicillin-resistant *Staphylococcus aureus* infection. *Antimicrob Agents Chemother* 2007; **51**: 4044–4048.

17 Duong M, Markwell S, Peter J, *et al*. Randomized, controlled trial of antibiotics in the management of community-acquired skin abscesses in the pediatric patient. *Ann Emerg Med* 2010; **55**: 401–407.

18 Schmitz G, Goodwin T, Singer A, *et al*. The treatment of cutaneous abscesses: comparison of emergency medicine providers' practice patterns. *West J Emerg Med* 2013; **14**: 23–28.

19 Talan DA, Mower WR, Krishnadasan A, *et al*. Trimethoprim–sulfamethoxazole versus placebo for uncomplicated skin abscess. *N Engl J Med* 2016; **374**: 823–832.

20 Talan DA, Moran GJ, Krishnadasan A, *et al*. Subgroup analysis of antibiotic treatment for skin abscesses. *Ann Emerg Med* 2018; **71**: 21–30.

21 Daum RS, Miller LG, Immergluck L, *et al*. A placebo-controlled trial of antibiotics for smaller skin abscesses. *N Engl J Med* 2017; **376**: 2545–2555.

22 Pulia MS, Schwei RJ, Patterson BW, *et al*. Effectiveness of outpatient antibiotics following surgical drainage of abscesses in reducing treatment failure. *J Emerg Med* 2018; **55**: 512–521.

23 Pulia M, Fox B. Clinical controversies: antibiotics should not be routinely prescribed following incision and drainage of uncomplicated abscesses. *Ann Emerg Med* 2019; **73**: 377–378.

24 Conway J, Friedman B. Adjuvant antibiotic therapy after incision and drainage of cutaneous abscesses. *Acad Emerg Med* 2020; **27**: 427–428.

25 Hua C, Urbina T, Bosc R, *et al*. Necrotising soft-tissue infections. *Lancet Infect Dis* 2023; **23**(3): e81–e94.

26 Rabuka CE, Azoulay LY, Kahn SR. Predictors of a positive duplex scan in patients with a clinical presentation compatible with deep vein thrombosis or cellulitis. *Can J Infect Dis* 2003; **14**(4): 210–214.

27 Bersier D, Bounameaux H. Cellulitis and deep vein thrombosis: a controversial association. *J Thromb Haemost* 2003; **1**(4): 867–868.

28 Arnold SR, Straus SE. Interventions to improve antibiotic prescribing practices in ambulatory care. *Cochrane Database Syst Rev* 2005; **2005**(4): CD003539.

29 Gonzales R, Bartlett JG, Besser RE, *et al*. Principles of appropriate antibiotic use for treatment of uncomplicated acute

bronchitis: background. *Ann Emerg Med* 2001; **37**(6): 720–727.
30 Centers for Disease Control and Prevention. Antibiotic prescribing and use: adult outpatient treatment recommendations. 2017. https://www.cdc.gov/antibiotic-use/clinicians/adult-treatment-rec.html. Accessed January 16, 2024.
31 Centor RM, Witherspoon JM, Dalton HP, *et al*. The diagnosis of strep throat in adults in the emergency room. *Med Decis Mak* 1981; **1**: 239–246.
32 Cooper RJ, Hoffman JR, Bartlett JG, *et al*. Principles of appropriate antibiotic use for acute pharyngitis in adults: background. *Ann Emerg Med* 2001; **37**(6): 711–719.
33 Wing A, Villa-Roel C, Yeh B, *et al*. Effectiveness of corticosteroid treatment in acute pharyngitis: a systematic review of the literature. *Acad Emerg Med* 2010; **17**(5): 476–483.
34 Rosenfeld RM, Piccirillo JF, Chandrasekhar SS, *et al*. Clinical practice guideline (update): adult sinusitis. *Otolaryngol Head Neck Surg* 2015; **152**(2 Suppl): S1–S39.
35 Gonzales R, Bartlett JG, Besser RE, *et al*. Principles of appropriate antibiotic use for treatment of acute respiratory tract infections in adults: background, specific aims, and methods. *Ann Emerg Med* 2001; **37**(6): 690–697.
36 De Sutter AIM, Eriksson L, van Driel ML. Oral antihistamine-decongestant-analgesic combinations for the common cold. *Cochrane Database Syst Rev* 2022; **1**(1): CD004976.
37 Salkind AR. Clostridium difficile: an update for the primary care clinician. *South Med J* 2010; **103**(9): 896–902.
38 Pituch H. Clostridium difficile is no longer just a nosocomial infection or an infection of adults. *Int J Antimicrob Agents* 2009; **33**(Suppl 1): S42–S45.
39 Guh AY, Mu Y, Winston LG, *et al*. Trends in U.S. burden of *Clostridioides difficile* infection and outcomes. *N Engl J Med* 2020; **382**: 1320.
40 Chitnis AS, Holzbauer SM, Belflower RM, *et al*. Epidemiology of community associated *Clostridium difficile* infection, 2009 through 2011. *JAMA Intern Med* 2013; **173**: 1359–1367.
41 Johnson S, Lavergne V, Skinner AM, *et al*. Clinical practice guideline by the Infectious Diseases Society of America (IDSA) and Society for Healthcare Epidemiology of America (SHEA): 2021 focused update guidelines on Management of *Clostridioides difficile* infection in adults. *Clin Infect Dis* 2021; **73**: e1029.
42 Bauer MP, van Dissel JT, Kuijper EJ. Clostridium difficile: controversies and approaches to management. *Curr Opin Infect Dis* 2009; **22**(6): 517–524.
43 Long B, Koyfman A. The emergency department diagnosis and management for urinary tract infection. *Emerg Med Clin N Am* 2018; **36**: 685–710.
44 Richards D, Toop L, Chambers S, *et al*. Response to antibiotics of women with symptoms of urinary tract infection but negative dipstick urine test results: double blind randomized controlled trial. *BMJ* 2005; **331**(7509): 143.
45 Cestari TF, Martignago BF. Scabies, pediculosis, bedbugs, and stinkbugs: uncommon presentations. *Clin Dermatol* 2005; **23**(6): 545–554.
46 Murray TS, Shapiro ED. Lyme disease. *Clin Lab Med* 2010; **30**(1): 311–328.
47 Nadelman RB, Nowakowski J, Fish D, *et al*. Prophylaxis with single-dose doxycycline for the prevention of Lyme disease after an Ixodes scapularis tick bite. *N Engl J Med* 2001; **345**(2): 79–84.
48 Centers for Disease Control and Prevention. Lyme disease prophylaxis after tick bite. In: *Tickborne Diseases of the United States: A Reference Manual for Healthcare Providers* (6e edn.). Washington, DC: U.S. Department of Health and Human Services; 2022: pp. 50–52.

CHAPTER 9
Headache

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Introduction

Headache is among the top six most common complaints in the emergency department (ED), accounting for over 4.6 million ED visits, or 3.3% of all visits, in the United States in 2021 [1]. While most headache complaints in urgent care settings are benign, it is crucial for healthcare providers to differentiate these from potentially serious causes requiring immediate diagnostic assessment and a higher level of care. Failing to identify severe or life-threatening causes of headaches can result in permanent neurologic deficits, vision loss, or even death.

According to the third edition of the International Classification of Headache Disorders (ICHD-3), headaches are usually categorized into "primary" or "secondary" [2]. Primary headaches have no known underlying cause and constitute the vast majority of headaches. These include migraine, tension-type headache, or trigeminal autonomic cephalalgia. Migraines often manifest unilaterally in adults but sometimes bilaterally in children and adolescents. They are typically gradual in onset, worsened by physical activity, and associated with symptoms like nausea, vomiting, photophobia, and/or phonophobia. Tension-type headaches, the most common type, are often bilateral, described as pressure or tightness, and may involve pericranial tenderness. Trigeminal autonomic cephalalgias, such as cluster headaches, are always unilateral and accompanied by cranial parasympathetic autonomic features. In cluster headaches, deep, unilateral pain is coupled with eye tearing, redness, a runny nose, and sweating [3]. These primary headaches can be treated symptomatically and referred to primary care for follow-up.

Secondary headaches, on the other hand, while less prevalent than primary headaches, stem from other medical conditions and may be linked to serious underlying disorders. These headaches often require complex diagnostics, such as neuroimaging, vital sign monitoring, and/or invasive procedures. It is therefore critical to distinguish between primary and secondary headaches. Examples of secondary headaches are shown in Box 9.1.

KEY FACT | **Although headaches are common, sometimes they are secondary to serious pathology that may require complex diagnostics and management.**

Pitfall | Failure to identify red flags associated with secondary headaches

Conducting a comprehensive history and physical examination is paramount in determining whether additional diagnostic evaluation is needed to investigate potential emergent causes of headaches. Certain risk factors or "red flags" identified during the history and examination may suggest the presence of secondary headaches.

For instance, a person older than 75 years presenting with headache has a 10-fold greater risk of adverse

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Box 9.1 Examples of causes of secondary headaches

- Traumatic head injury
- Acute stroke, ischemic or hemorrhagic
- Unruptured vascular malformation
- Giant cell arteritis
- Cervical carotid or vertebral artery dissection
- Cerebral venous and dural sinus thrombosis
- Reversible cerebral vasoconstriction syndrome
- Genetic vasculopathy
- Hypertensive emergency
- Idiopathic intracranial hypertension
- Intracranial neoplasm
- Neurosarcoidosis
- Carbon monoxide poisoning
- Medication overuse headache
- Substance withdrawal
- Meningitis or meningoencephalitis
- Systemic infection
- Hypoxia or hypercapnia
- Acute angle-closure glaucoma

Source: Adapted and summarized from ICHD-3 [2].

pathology compared to a person younger than 50 years. A headache with "sudden onset" is 97% sensitive for subarachnoid hemorrhage (SAH) due to an aneurysm. The presence of either fever, neck stiffness, or altered mental status is present in almost all patients with meningitis [4]. Additional examples of red flags are shown in Box 9.2. Many secondary causes of headache require emergency evaluation and a higher level of care. These include suspicion for central nervous system (CNS) infection, acute hemorrhage, stroke, cerebral sinus thrombosis, hypertensive crisis, and carbon monoxide (CO) poisoning, among others.

On the other hand, it is important to note that serious causes of headache remain relatively rare in the urgent care setting. Even with the presence of red flags, the positive predictive value remains low. In one prospective study where patients with nonacute headache underwent neuroimaging, only 2.1% of these patients were found to have an abnormality on imaging, compared to 0.3% of the total study population [5]. A normal neurological exam reduces the risk of malignancy by half, from 1 in 20 to 1 in 40 [4]. Therefore, careful history and physical examination alone may suggest that the patient does not need further emergency testing.

KEY FACT | **History and physical examination are the keys to understanding who requires aggressive diagnostic studies to rule out emergency causes of headache.**

Pitfall | Assuming that a patient with a history of headaches has a benign cause for their headache

Headaches are extremely common, with a lifetime prevalence of 96% [7]. Active headache disorder is estimated to be present in 52% of the global population, with migraine in 14% and tension-type headache in 26% [8]. Therefore it can be assumed that most patients who present for evaluation of a headache have suffered one in the past.

Many patients with primary headache disorder may suffer from recurrent headaches. However, a change in the pattern or quality of the headache raises the possibility of secondary causes. Because they are common and usually benign, it is imperative that providers are vigilant in considering secondary causes for headache and that they test, manage, and refer patients appropriately.

KEY FACT | **Headaches are nearly ubiquitous in the population, and a history of prior headaches does not rule out worrisome secondary causes of headache.**

Pitfall | Assuming that a headache is benign because it responds to treatment

A favorable response to treatment for an acute headache does not necessarily suggest a benign cause. A systematic review of 18 journal articles examining treatment response of headaches suggested that a subset of patients who reported significant or complete resolution of headache from treatment ultimately had a delay in real diagnosis by hours to weeks [9]. Response to triptans, anti-inflammatories, or analgesics can occur with any cause of headache, primary or secondary, and must not be used to exclude serious etiology. Serious causes of headaches can also present as intermittent pain in nature, thus providing false reassurance to the treating provider [9].

Box 9.2 Historical or physical examination features that may suggest a serious secondary cause of headache, based on SNNOOP10 criteria

Clinical features	Possible secondary headache causes
Systemic symptoms including fever	Infection or nonvascular intracranial disorders
Neoplasm in history	Central nervous system neoplasm
Neurologic deficit or dysfunction	Headaches attributed to vascular/nonvascular intracranial disorders; brain abscess
Onset of headache is sudden or abrupt	Subarachnoid hemorrhage
Older age (after 50 years)	Giant cell arteritis; neoplasms; other intracranial disorders
Pattern change or recent onset of headache	Neoplasms; headaches attributed to vascular/nonvascular intracranial disorders
Positional headache	Intracranial hypertension or hypotension
Precipitated by sneezing, coughing, or exercise	Posterior fossa malformations
Papilledema	Neoplasms and other nonvascular intracranial disorders; intracranial hypertension
Progressive headache and atypical presentations	Neoplasms; other nonvascular intracranial disorders
Pregnancy or puerperium	Cranial or cervical vascular disorders; preeclampsia; cerebral sinus thrombosis
Painful eye with autonomic features	Pathology in posterior fossa, pituitary region, or cavernous sinus; ophthalmic causes
Posttraumatic onset of headache	Acute and chronic posttraumatic headache; subdural hematoma and other vascular disorders
Pathology of the immune system such as HIV	Opportunistic infections
Painkiller overuse or new drug at onset of headache	Medication overuse headache; drug incompatibility

Source: Adapted from Do *et al*. [6].

KEY FACT | **A favorable response to symptomatic treatment does not rule out secondary causes of headache.**

Pitfall | Relying on noncontrast head computed tomography alone to rule out subarachnoid hemorrhage in all patients

Noncontrast head computed tomography (CT) is routinely performed in patients with headache exhibiting concerning features for SAH. Several studies indicate that a negative noncontrast head CT performed within 6 hours of symptom onset is highly effective in excluding SAH [10]. However, over time, as blood products in the brain undergo changes and become isodense with brain tissue, they may become challenging to distinguish on CT scans, which can occur hours to weeks after symptom onset [11]. Therefore, the sensitivity of noncontrast head CTs to detect SAHs diminishes with time.

Patients who present more than six hours after the onset of symptoms with a persistent concern for SAH should undergo further evaluation. Traditionally, lumbar punctures have been performed due to their excellent sensitivity in ruling out SAH [12]. However, lumbar punctures have limitations, including a low diagnostic yield, high incidence of traumatic taps, time constraints, patient discomfort, and the potential for inducing post-lumbar puncture headaches [13].

An alternative approach is to utilize CT followed by CT angiography (CTA). CTA has shown high accuracy in diagnosing cerebral aneurysms in patients with nontraumatic acute SAH, with one study suggesting a sensitivity of 99.3% [14]. However, CTA is only able to accurately diagnose aneurysms, not acute bleeding. Thus, a positive finding of an aneurysm on CTA may not always suggest acute bleeding, which could lead to unnecessary downstream interventions. Additionally, CTA exposes patients to additional radiation. Deferring a lumbar puncture may also occasionally result in missing alternative diagnoses like meningitis or increased intracranial pressure [15].

The benefits and risks of each strategy should be discussed with the patient through shared decision making. Nonetheless, noncontrast head CT alone is likely

inadequate to rule out SAH in patients who present with concerning symptoms more than six hours after onset.

KEY FACT | **For patients with a history suggestive of subarachnoid hemorrhage and symptoms >6 hours, a normal noncontrast computed tomography (CT) scan warrants further evaluation with lumbar puncture or CT angiography.**

Pitfall | Failure to consider meningitis in the afebrile patient

Meningitis is a type of CNS infection that should be considered in patients of all ages presenting to the acute care setting with headaches. The overall incidence of bacterial meningitis specifically has decreased over the last few decades due to the introduction of conjugate vaccines and other preventive measures; however, it still carries substantial morbidity [16, 17]. If missed, bacterial meningitis is nearly always fatal [18].

The classic triad of acute bacterial meningitis is fever, neck stiffness, and mental status change. However, the triad is only observed in 44% of adult patients with confirmed bacterial meningitis. The most common symptom is severe headache (87% of patients), followed by neck stiffness (83%), fever (77%), and mental status changes (69%) [19]. Almost all (95%) have at least two of the four symptoms. It is important to note, then, that approximately one-fourth of patients with confirmed bacterial meningitis will *not* have a fever at presentation. Atypical presentations can be even more frequent in patients who are immunocompromised [20].

Evaluation of meningitis includes obtaining CT head noncontrast imaging (if indicated to evaluate for space-occupying lesion) and lumbar puncture to assess for infection. However, if there is a high clinical suspicion for meningitis, obtaining these studies should not delay treatment. Delaying antibiotics in bacterial meningitis is linked with increased mortality [21]. Empiric intravenous (IV) antibiotics and steroids should be initiated promptly in the urgent care setting, even if the CT and lumbar puncture have not yet been obtained. The patient should then be transferred to a high level of care, where further studies can be obtained if necessary.

KEY FACT | **Patients with meningitis may be afebrile.**

Pitfall | Failure to note high blood pressure in the pregnant or postpartum patient with headache

If the acute care clinician encounters a pregnant or postpartum patient with a headache, they must pay particular attention to the blood pressure. A blood pressure reading of 140/90 mmHg or higher should prompt further evaluation for preeclampsia. Preeclampsia is a major contributor to maternal morbidity and mortality. Risk factors include first pregnancy, advanced maternal age, multiple medical comorbidities, multigestational pregnancy, and personal or family history of preeclampsia [22]. The burden of disease in the United States is seen particularly in African American women, with higher prevalence and threefold higher mortality rate compared to white women [23].

Preeclampsia is defined classically as hypertension and proteinuria occurring after 20 weeks of gestation until six weeks postpartum. Physical exam may demonstrate peripheral edema. In severe preeclampsia the patient may also develop signs of end-organ dysfunction, including vision changes, mental status changes, dyspnea, oliguria, and abdominal pain [24]. HELLP syndrome may also be seen in these patients, defined by hemolysis, elevated liver enzymes, and low platelets. See the full diagnostic criteria in Box 9.3.

Box 9.3 Diagnostic criteria for preeclampsia

Hypertension

- Blood pressure 140/90 mmHg or greater on two occasions at least 4 hours apart *or*
- Blood pressure greater than 160/110 mmHg on one occasion

Plus:
Proteinuria

- 300 mg or more in 24 h urine collection *or*
- Protein/creatinine ratio of at least 0.3 mg/dl *or*
- Protein 2+ on dipstick

Or in absence of proteinuria

- Thrombocytopenia: platelet count <10,000
- Elevation of liver enzymes twice normal
- New renal insufficiency: creatinine greater than 1.1 or doubling of baseline
- Pulmonary edema
- New-onset mental status disturbances

If left untreated preeclampsia can progress to eclampsia, which is characterized by seizures. Treatment for preeclampsia therefore includes IV magnesium sulfate load followed by a maintenance infusion for seizure prevention. Prompt blood pressure control should be initiated with either IV labetalol or hydralazine. Definitive therapy in pregnant patients is delivery of the fetus. Whether pregnant or postpartum, a patient with preeclampsia will need emergent obstetric consultation and transfer for further management [24].

KEY FACT | **Always consider preeclampsia in pregnant and postpartum patients presenting with headaches.**

Pitfall | Failure to identify headaches associated with increased intracerebral pressure

Idiopathic intracranial hypertension (IIH), otherwise known as pseudotumor cerebri, is defined by clinical manifestations of elevated intracranial pressure. These include headache, visual disturbances, papilledema, and an elevated opening pressure on lumbar puncture.

IIH is seen primarily in obese women ages 20–44 years [24]. The most common symptom is headache (85–92% of patients) [25]. Although variable, the headaches of IIH are often described as quite severe and may worsen with postural changes. Patients may also have back pain, pain behind the eyes, and pulsatile tinnitus. They will frequently have visual complaints along with their headache, such as transient visual obscurations or diplopia. Patients with a severe headache and new visual symptoms should raise concern for possible IIH.

The key finding on physical examination is papilledema, which is present in the vast majority (94%) of cases [26]. The patient's neurological examination is often normal, but cranial nerve deficits, particularly the abducens nerve in patients with diplopia, can occur due to increased intracranial pressure [27].

If the acute care practitioner is considering IIH, then brain imaging and lumbar puncture with opening pressure measurement should be obtained. The patient will also need ophthalmologic evaluation. Neuroimaging is important in the evaluation of IIH primarily to exclude other causes of the patient's headache. Magnetic resonance imaging (MRI) is the preferred imaging modality, but CT is an acceptable starting point if there is no access to MRI. If these studies cannot be obtained at one's particular institution, the patient should be transferred to a higher level of care, since undiagnosed IIH can lead to permanent vision loss.

KEY FACT | **Delayed diagnosis of idiopathic intracranial hypertension can lead to permanent vision loss.**

Pitfall | Failure to consider cerebral venous thrombosis as the cause of headache

Cerebral venous thrombosis, or the formation of a blood clot in the venous drainage system of the brain, accounts for up to 1% of all strokes and is an important cause of headaches [28, 29]. It occurs more commonly in younger adult patients and frequently goes unrecognized. Risk factors are similar to those associated with venous thromboembolism in general. These include inherited forms of thrombophilia as well as acquired hypercoagulable states, particularly pregnancy and postpartum, oral contraceptive use, and cancer [28, 30–32]. It is three times more common in females [30].

Presentation for cerebral venous thrombosis is variable, contributing to the difficulty in diagnosis. Headache is the most common symptom of cerebral venous thrombosis and is usually progressive over days [28, 32]. Patients may present with findings seen from increased intracranial pressure due to impaired venous drainage, such as papilledema or diplopia. They may also exhibit neurologic deficits due to brain injury from ischemia or hemorrhage. However, up to 25% of patients will have isolated headache without any other associated neurologic findings [28]. Therefore, clinicians should have a high index of suspicion for this diagnosis in anyone with new headaches and a prothrombotic risk factor.

Cerebral venous thrombosis is often missed on noncontrast CT. If there is clinical suspicion for this diagnosis, the test of choice is CT venography (CTV) or magnetic resonance venography (MRV). Once the diagnosis is made, anticoagulation therapy with unfractionated heparin or low molecular weight heparin should be initiated [28, 30]. The patient should be transferred to a higher level of care for further management.

KEY FACT | **In patients with prothrombotic risk factors, computed tomography venography or magnetic resonance venography should be considered to evaluate for cerebral venous thrombosis.**

Pitfall | **Not evaluating for extracranial causes of headache**

Many extracranial secondary causes for headaches exist. Three important causes that urgent care clinicians should consider include temporal arteritis, acute angle-closure glaucoma, and exposures such as CO. A thorough history and physical examination are key for identifying these disease processes.

Temporal arteritis (TA), also called giant cell arteritis, is a systemic vasculitis involving medium-sized and large arteries. TA occurs in patients over 50 years old, is more common in females, and is associated with a history of polymyalgia rheumatica. Most patients with TA present with headaches. These are typically in the temporal region and are often described as a change from any baseline headache. Other symptoms include jaw claudication, scalp tenderness, and constitutional symptoms such as weight loss or low-grade fever. The most feared complication of TA is vision loss due to ischemic optic neuropathy [33].

Examination may reveal a prominent temporal artery, decreased temporal pulse, or temporal artery tenderness [33]. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) should be ordered and, although nonspecific, are often markedly elevated. In biopsy-proven TA, one study found that 4% of patients had normal ESR and CRP [34]. The ultimate diagnosis is made by temporal artery biopsy. If clinical concern is high, steroids should be initiated early to avoid permanent vison loss. While awaiting arrangement of urgent biopsy, 40–60 mg/day of prednisone should be initiated for a patient with no visual symptoms. A patient with visual symptoms will need 1 g daily IV methylprednisolone for the first three days and will therefore likely need a higher level of care [33, 35].

Acute angle-closure glaucoma is an ocular emergency caused by an obstruction of aqueous humor drainage in the eye leading to a rapid increase in intraocular pressure (IOP). If left untreated, it will progress to permanent vision loss. Patients usually present with sudden onset of unilateral eye pain or headache. They may also experience blurred vision, nausea, and vomiting. It occurs more typically in females and older patients [36], commonly at night or in environments with lower light levels [37].

Physical examination of the eye will demonstrate a fixed, mid-dilated pupil with surrounding conjunctival injection. Visual acuity should be performed and measurement of IOP is crucial. IOPs will be above 20 mmHg in the affected eye but often are between 40 and 70 mmHg [24]. Once acute angle-closure glaucoma is identified, emergent consultation from ophthalmology should be obtained. Treatment to rapidly decrease the IOP should be initiated by the urgent care clinician.

Treatment regimens vary but involve a combination of medications, including a topical beta-blocker, a topical alpha-2 agonist, oral or IV acetazolamide, and subsequently topical pilocarpine once IOPs are below 40 mmHg [24]. IOP in the affected eye should be assessed every hour to evaluate treatment efficacy [24]. The patient may require transfer, depending on the institution's capability and access to ophthalmology. The definitive treatment is iridotomy (creating a hole in the peripheral iris), performed by ophthalmology [36].

Finally, consider CO poisoning as the cause of a patient's headache. CO poisoning occurs in 50,000 people per year in the United States [38]. CO is formed from incomplete combustion, with common forms of exposure including house fires, use of fuel-burning equipment with improper ventilation, and exhaust from gas vehicles [38, 39]. Symptoms of severe CO poisoning include loss of consciousness, confusion, and cardiopulmonary failure. However, symptoms are likely to be much more subtle for patients evaluated in the urgent care setting. In fact, the most common symptom that patients with CO poisoning report is headache, followed by other nonspecific symptoms such as dizziness, fatigue, and nausea [39, 40]. A vital question to ask in the review of symptoms is: "Does anyone around you have similar symptoms?" Headaches occurring in groups of people from the same environment should prompt consideration of CO poisoning.

Suspected CO poisoning should be evaluated by blood gas testing to measure the carboxyhemoglobin level. If this cannot be performed in the urgent care setting, the patient should be referred for this testing emergently. If confirmed, treatment with high-flow oxygen therapy and hyperbaric oxygen therapy may be indicated. Identifying CO poisoning can be lifesaving not only for the patient but also for other exposed household members or coworkers.

KEY FACT | **There are serious extracranial secondary causes for headache, including temporal arteritis, acute angle-closure glaucoma, and carbon monoxide poisoning.**

Pearls for improving patient outcomes

• Although headaches are common, sometimes they are secondary to serious pathology that may require complex diagnostics and management.
• History and physical exam are the keys to understanding who requires aggressive diagnostic studies or management for their headaches.
• Headaches are nearly ubiquitous in the population, and a history of prior headaches does not rule out worrisome secondary causes of the current headache.
• Favorable response to symptomatic treatment does not rule out secondary causes of headache.
• For patients with history suggestive of subarachnoid hemorrhage and symptoms >6 hours, a normal noncontrast computed tomography (CT) scan warrants further evaluation with lumbar puncture or CT angiography.
• Patients with meningitis may be afebrile.
• Always consider preeclampsia in pregnant and postpartum patients presenting with headaches.
• Delayed diagnosis of increased intracranial hypertension can lead to permanent vision loss.
• In patients with prothrombotic risk factors, CT venography or magnetic resonance venography should be considered to evaluate for cerebral venous thrombosis.
• There are serious extracranial secondary causes for headache, including temporal arteritis, acute angle-closure glaucoma, and carbon monoxide exposure.

References

1 National Center for Health Statistics. National Hospital Ambulatory Medical Care Survey: 2021 Emergency Department Summary Tables. 2021. https://www.cdc.gov/nchs/data/nhamcs/web_tables/2021-nhamcs-ed-web-tables-508.pdf. Accessed July 22, 2024.
2 Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. *Cephalalgia* 2018; **38**(1): 1–211. https://doi.org/10.1177/0333102417738202.
3 Wippold FJ II, Whealy MJ, Kaniecki RG. Evaluation of headache in adults. 2023. https://www.uptodate.com/contents/evaluation-of-headache-in-adults. Accessed October 12, 2023.
4 Filler L, Akhter M, Nimlos P. Evaluation and management of the emergency department headache. *Semin Neurol* 2019; **39**(1): 20–26.
5 Clarke CE, Edwards J, Nicholl DJ, *et al*. Imaging results in a consecutive series of 530 new patients in the Birmingham Headache Service. *J Neurol* 2010; **257**(8): 1274–1278.
6 Do TP, Remmers A, Schytz HW, *et al*. Red and orange flags for secondary headaches in clinical practice: SNNOOP10 list. *Neurology* 2019; **92**(3): 134–144.
7 Rizzoli P, Mullally WJ. Headache. *Am J Med* 2018; **131**(1): 17–24.
8 Stovner LJ, Hagen K, Linde M, *et al*. The global prevalence of headache: an update, with analysis of the influences of methodological factors on prevalence estimates. *J Headache Pain* 2022; **23**(1): 34.
9 Pope JV, Edlow JA. Favorable response to analgesics does not predict a benign etiology of headache. *Headache* 2008; **48**(6): 944–950.
10 Carpenter CR, Hussain AM, Ward MJ, *et al*. Spontaneous subarachnoid hemorrhage: a systematic review and meta-analysis describing the diagnostic accuracy of history, physical examination, imaging, and lumbar puncture with an exploration of test thresholds. *Acad Emerg Med* 2016; **23**(9): 963–1003.
11 Chakeres DW, Bryan RN. Acute subarachnoid hemorrhage: in vitro comparison of magnetic resonance and computed tomography. *AJNR Am J Neuroradiol* 1986; **7**(2): 223–228.
12 Perry JJ, Spacek A, Forbes M, *et al*. Is the combination of negative computed tomography result and negative lumbar puncture result sufficient to rule out subarachnoid hemorrhage? *Ann Emerg Med* 2008; **51**(6): 707–713.
13 Engelborghs S, Niemantsverdriet E, Struyfs H, *et al*. Consensus guidelines for lumbar puncture in patients with neurological diseases. *Alzheimers Dement (Amst)* 2017; **8**: 111–126.
14 El Khaldi M, Pernter P, Ferro F, *et al*. Detection of cerebral aneurysms in nontraumatic subarachnoid haemorrhage: role of multislice CT angiography in 130 consecutive patients. *Radiol Med* 2007; **112**(1): 123–137.
15 American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Acute Headache, Godwin SA, Cherkas DS, *et al*. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute headache. *Ann Emerg Med* 2019; **74**(4): e41–e74.
16 Castelblanco RL, Lee M, Hasbun R. Epidemiology of bacterial meningitis in the USA from 1997 to 2010: a population-based observational study. *Lancet Infect Dis* 2014; **14**(9): 813–819.
17 Thigpen MC, Whitney CG, Messonnier NE, *et al*. Bacterial meningitis in the United States, 1998–2007. *N Engl J Med* 2011; **364**(21): 2016–2025.
18 Swartz MN. Bacterial meningitis—a view of the past 90 years. *N Engl J Med* 2004; **351**(18): 1826.
19 Van de Beek D, de Gans J, Spanjaard L, *et al*. Clinical features and prognostic factors in adults with bacterial meningitis. *N Engl J Med* 2004; **351**(18): 1849–1859.
20 Pruitt AA. Central nervous system infections in immunocompromised patients. *Curr Neurol Neurosci Rep* 2021; **21**(7): 37.

21 Bodilsen J, Dalager-Pedersen M, Schønheyder HC, *et al*. Time to antibiotic therapy and outcome in bacterial meningitis: a Danish population-based cohort study. *BMC Infect Dis* 2016; **16**: 392.
22 Gestational Hypertension and Preeclampsia. ACOG Practice Bulletin, Number 222. *Obstet Gynecol* 2020; **135**(6): p237–p260.
23 Zhang M, Wan P, Ng K, *et al*. Preeclampsia among African American pregnant women: an update on prevalence, complications, etiology, and biomarkers. *Obstet Gynecol Surv* 2020; **75**(2): 111–120.
24 Cydulka RK, Fitch MT, Joing SA, *et al*. *Tintinalli's Emergency Medicine Manual* (8th edn.). New York: McGraw Hill; 2018.
25 Peng KP, Fuh JL, Wang SJ. High-pressure headaches: idiopathic intracranial hypertension and its mimics. *Nat Rev Neurol* 2012; **8**: 700–710.
26 Digre KB, Nakamoto BK, JEA W, *et al*. A comparison of idiopathic intracranial hypertension with and without papilledema. *Headache* 2009; **49**: 185–193.
27 Wall M, Kupersmith MJ, Kieburtz KD, *et al*. The idiopathic intracranial hypertension treatment trial: clinical profile at baseline. *JAMA Neurol* 2014; **71**(6): 693–701.
28 Saposnik G, Barinagarrementeria F, Brown RD Jr, *et al*. Diagnosis and management of cerebral venous thrombosis. *Stroke* 2011; **42**(4): 1158–1192.
29 Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol* 2007; **6**: p162–p170.
30 Ulivi L, Squitieri M, Cohen H, *et al*. Cerebral venous thrombosis: a practical guide. *Pract Neurol* 2020; **20**: 356–367.
31 Duman T, Uluduz D, Midi I, *et al*. A multicenter study of 1144 patients with cerebral venous thrombosis: the VENOST study. *J Stroke Cerebrovasc Dis* 2017; **26**: 1848–1857.
32 Ferro JM, Canhão P, Stam J, *et al*. Prognosis of cerebral vein and dural sinus thrombosis: results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT). *Stroke* 2004; **35**: 664–670.
33 Salvarani C, Cantini F, Hunder GG. Polymyalgia rheumatica and giant-cell arteritis. *Lancet* 2008; **372**: 234.
34 Kermani TA, Schmidt J, Crowson CS, *et al*. Utility of erythrocyte sedimentation rate and C-reactive protein for the diagnosis of giant cell arteritis. *Semin Arthritis Rheum* 2012; **41**(6): p866–p871.
35 Dasgupta B, Borg FA, Hassan N, *et al*. BSR and BHPR guidelines for the management of giant cell arteritis. *Rheumatology* 2010; **49**(8): 1594–1597.
36 Prum BE, Herndon LW Jr, Moroi SE, *et al*. Primary angle closure preferred practice pattern(®) guidelines. *Ophthalmology* 2016; **123**(1): P1.
37 Leibowitz HM. The red eye. *N Engl J Med* 2000; **343**(5): 345.
38 Rose JJ, Wang L, Xu Q, *et al*. Carbon monoxide poisoning: pathogenesis, management, and future directions of therapy. *Am J Respir Crit Care Med* 2017; **195**(5): 596–606.
39 Kinoshita H, Türkan H, Vucinic S, *et al*. Carbon monoxide poisoning. *Toxicol Rep* 2020; **7**: 169–173.
40 Shin M, Bronstein AC, Glidden E, *et al*. Morbidity and mortality of unintentional carbon monoxide poisoning: United States 2005 to 2018. *Ann Emerg Med* 2023; **81**(3): 309.

CHAPTER 10
The Evaluation and Management of Back Pain

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Introduction

Back pain is an extremely common complaint among patients presenting to urgent care centers and emergency departments (EDs). Globally, low back pain is the most common musculoskeletal problem, with an age-standardized point prevalence of 8.20% and a higher prevalence in female patients. Low back pain was the leading cause of years lived with disability in both 1990 and 2017 out of all conditions studied in the Global Burden of Disease study [1]. The annual direct care cost for back pain is estimated to be $12.2–90.6 billion in the United States [2]. These figures do not include the indirect costs of lost work days or disability payments, estimated to be $7.4–28.2 billion per year [3]. In 2013, low back and neck pain was ranked the third highest condition for personal healthcare spending in the United States, trailing only diabetes and ischemic heart disease [4].

Pitfall | **Failure to recognize the "red flags" in the medical history or physical exam that suggest a serious cause for a patient's back pain**

Most cases of back pain do not have a discernible cause, but they have a predictable course. A subset of patients is at risk for chronic pain and disability, and in another subset a serious life-threatening or devastating neurologic disease will develop if appropriate treatment is not administered. Identifying the patients at risk for serious morbidity and mortality is paramount in the evaluation of back pain. Given the frequency with which musculoskeletal back pain is seen in the acute care setting, it is easy to become complacent with the evaluation. This is most likely to occur when evaluating patients who have a history of chronic back pain, as an acute exacerbation of the pain is the most likely cause of their presentation. However, despite chronicity of pain, it is always important to consider that a more serious disease could be present.

The evaluation of all patients with back pain should include a comprehensive history and physical exam. Describing how to conduct a complete physical exam is beyond the scope of this chapter, but the key factors that should be documented are highlighted here. The history should specifically cover the "red flags" in Box 10.1. Patients with any of these signs and symptoms should undergo additional diagnostic testing as indicated.

All patients should be undressed for the examination. This allows direct visualization of rashes (e.g. herpes zoster); asymmetric muscle development or atrophy; scars that indicate previous surgical repairs or trauma; and evidence of recent trauma, such as lacerations, abrasions, or ecchymosis. It also permits a more thorough sensory exam. The physical examination should consist of formal and functional strength testing.

Formal testing typically consists of the patient resisting against the examiner and is graded on a scale of 1–5. However, because of pain, many patients are unable or unwilling to cooperate fully with strength testing. Having patients walk, lift up on their toes, rock back on their heels, and squat allows evaluation of the strength in their quadriceps, hamstrings, and dorsiflexors. If the

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Box 10.1 Red flags in patient history and physical examination

Red flags: history	Red flags: physical
• Age <18 or >50 years • Pain lasting more than six weeks • History of cancer • Fever • Night sweats • Unexplained weight loss • Recent bacterial infection • Intravenous drug abuse • Immunocompromised state (e.g. HIV infection, immunosuppressive medication, chronic steroid use) • Major trauma • Minor trauma in the elderly • Night pain or pain that increases when supine	• Fever • Bowel or bladder incontinence • Saddle anesthesia • Decreased or absent anal sphincter tone • New neurologic deficit

patient is unable to raise one leg, the examiner should note whether the patient is pushing down with the other leg. If no contralateral effort is being exerted (i.e. the patient is not pushing down), then the patient either is not fully cooperating with the examination or has bilateral leg weakness.

The patient's gait should be observed. Ideally, when possible, the examiner should watch the patient's gait as they walk to the examination room. It is not unusual for the gait to be slower and less natural when patients know they are being observed. Special attention should be directed toward ensuring that the patient does not have foot drop, is not dragging a leg, and has a normal swing phase of the gait when all the weight is on a single leg. With the patient in a standing position, their range of motion in anterior flexion, posterior flexion, and lateral flexion should be assessed.

If the patient complains of urinary or stool incontinence, or if the physical examination yields findings indicative of incontinence, a rectal examination checking sphincter tone should be performed. If the patient has urinary incontinence, a bladder scan should be requested or a postvoid residual measured to exclude a neurogenic bladder. Incontinence can be caused by overflow incontinence from the inability to drain the bladder completely.

Pediatric back pain

Historically, it has been taught that back pain in children and adolescents is rare; however, the true incidence is not known, as many children with this pain do not seek medical care. One study in 1998 that surveyed 5000 children found that 7% of 12-year-olds reported at least one episode of back pain [5]. Recent literature suggests that a relatively high number of children do experience back pain [6]. As in adults, an identifiable cause of the pain often is not evident. Davis and Williams modified the traditional red flags to address the specific disease processes that can occur in the pediatric population (Box 10.2) [6]. The physical examination in the child is like that in adults, though special care should be taken to look for scoliosis and kyphosis. A lower-extremity examination is also needed to ensure that the child is not experiencing referred pain from the knee or hip.

KEY FACT | **Recent literature suggests that a relatively large number of children do experience back pain. As in adults, an identifiable cause of the pain often is not evident.**

As in adults, children should have an assessment of their forward flexion. Schober's measurement

Box 10.2 Red flags in history and physical examination in children

Red flags: history	Red flags: physical
• Prepubertal children, especially <5 years • Functional disability • Duration >4 weeks • Recurrent or worsening pain • Early-morning stiffness and/or gelling • Night pain • Fever, weight loss, malaise • Postural changes: kyphosis or scoliosis • Limp or altered gait	• Fever, tachycardia • Weight loss, bruising, lymphadenopathy, or abdominal mass • Altered spine shape or mobility • Vertebral or intervertebral tenderness • Limp or altered gait • Neurologic symptoms • Bladder or bowel dysfunction

Source: Reproduced from Davis and Williams [6] with permission from the BMJ Publishing Group.

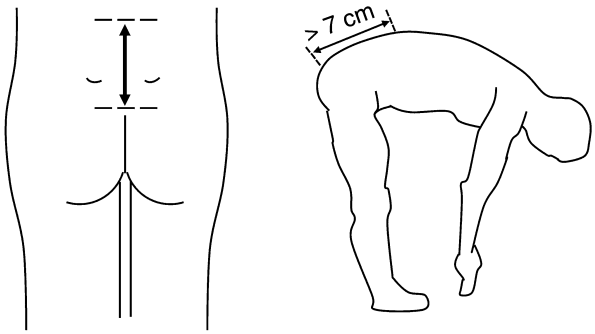

Figure 10.1 Schober's measurement. Place a mark 10 cm above and 5 cm below the level of the sacral dimples. This distance is measured and the patient is then asked to touch their toes. The distance between the marks is remeasured at the patient's maximum reach. A problem with lumbar mobility is suggested if the distance between the marks does not increase by more than 7 cm.

(see Figure 10.1) is obtained by placing a mark 10 cm above and 5 cm below the level of the sacral dimples. This distance is measured and the patient is then asked to touch their toes. The distance between the marks is measured again at the patient's maximum reach. A problem with lumbar mobility is suggested if the distance between the marks does not increase by more than 7 cm. Children with scoliosis should have a determination of whether it is fixed or reversible. Fixed scoliosis does not change with movements and is caused by a structural anomaly. Reversible scoliosis is typically related to posture or an abnormality of the pelvis or leg (e.g. a leg length discrepancy).

Evaluation

There is no standard evaluation that is required in patients with back pain. In the majority of cases, regardless of the patient's age, laboratory studies—complete blood count (CBC), urinalysis, blood cultures, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR)—radiographs, and magnetic resonance imaging (MRI) do not alter the management of the patient and only increase the cost of care. Radiographs should be ordered in cases of trauma, for the elderly when there is concern for compression fractures or cancer, and for children in whom a congenital anomaly or occult trauma may be present. MRI scans often identify pathology that may not be actually responsible for the patient's pain. In a study by Jensen *et al.*, 52% of completely asymptomatic individuals had at least one herniated nucleus pulposus (HNP) on MRI [7]. Similar results have been found in other studies, so it has become clear that HNPs are often found but are not necessarily the cause of the patient's back pain. In fact, MRI is best used to determine whether the HNP is compressing any neural structures. MRI is the preferred test if there is concern for cancer, infection, disk pathology, or spinal stenosis. Computed tomography (CT) scans are better for bony detail and are preferred if a fracture is suspected.

KEY FACT | **In the vast majority of cases, laboratory studies and imaging do not alter the management and only increase the cost of care.**

Treatment

The treatment of low back pain should be aimed at returning the patient to work and the usual activity level as quickly as possible. Most patients improve within six weeks regardless of the treatment rendered. Patients should be encouraged to return to light activity. Bed rest should be discouraged. A recent Cochrane review revealed moderate evidence that patients experience pain relief and a return to functional status when they stay active [8].

KEY FACT | **Most patients improve within weeks regardless of the treatment rendered.**

The preferred drugs for pain control are nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen, which have been shown to be as effective as muscle relaxants and opioids [9, 10]. Opioids and muscle relaxants are associated with more severe side effects and should be used only when absolutely needed and for the shortest time possible. The recently published OPAL trial showed that short courses of opioids are associated with increased pain at 26 and 52 weeks when compared to placebo [11]. Despite the data against opioids, an analysis of the National Hospital Ambulatory Medical Care Survey showed that two-thirds of ED patients who present with back pain are prescribed opioids, which are prescribed more often than NSAIDs [12]. There is no proven benefit to glucocorticoids in treating back pain and sciatica [9, 13]. A recent Cochrane review showed that steroids do improve pain in radicular back pain over nonradicular back pain, but this improvement was less than the minimal clinically significant difference [13]. Steroids are not recommended because of their side-effect profile, which includes an increased risk of infection, gastrointestinal bleeding, and hyperglycemia.

Exercise is one of the few treatment modalities shown to be effective for chronic low back pain [14]. Patients with chronic pain and those who are not responding to conservative therapy should be referred to physical therapy and, if feasible, patients preferred to see a physical therapist while in the ED [15]. After six weeks of conservative treatment, and if physical therapy is ineffective, patients should be referred to a back pain specialist for further diagnostic tests (e.g. MRI), consideration for epidural corticosteroid injections, or surgery.

KEY FACT | **Exercise is one of the few treatment modalities shown to be effective for chronic low back pain.**

Specific disease processes

Pitfall | Failure to consider the diagnosis of spinal epidural abscess because the patient does not have a fever or neurologic deficit

Epidural abscess

Spinal epidural abscesses (SEAs) are rare, with an estimated incidence of 1 in 10,000 hospital admissions. They have been increasing in frequency over the past 25 years. Risk factors for SEA are listed in Box 10.3. The classic triad of fever, back pain, and neurologic deficit is seen in less than 15% of patients at the time of initial presentation, and only 50% of this group are initially febrile [16]. This makes the diagnosis of SEA challenging. Half of the cases of SEA are misdiagnosed on the initial visit. The diagnosis should be considered in patients with new onset of back pain and risk factors or with back pain associated with fever, chills, night sweats, weight loss, or any neurologic deficit.

Box 10.3 Risk factors for spinal epidural abscess

- Diabetes
- End-stage renal disease
- Septicemia
- HIV infection
- Malignancy
- Morbid obesity
- Long-term glucocorticoid use
- Intravenous drug abuse
- Alcoholism
- Infection at a distal site
- Indwelling catheters
- History of spinal surgery

Source: Adapted from Tompkins *et al*. [18].

Unfortunately, laboratory tests alone cannot make the diagnosis. Neither the total white blood cell (WBC) count nor the leukocyte percentage is helpful in the diagnosis. Several studies have shown that the WBC count is greater than 12,000 cells/mm^3 in only 60% of cases [17]. CRP and ESR can be elevated but are not specific for SEA and are best used to follow the patient's response to therapy. Blood cultures are positive in only 60% of patients. Plain radiographs can visualize the very late signs associated with osteomyelitis or foreign bodies [18]. MRI with gadolinium is the only test that is sensitive and specific enough to make the diagnosis. SEAs are often found at multiple noncontiguous spinal levels (known as skip lesions), which can affect operative management and long-term outcomes. Therefore, if SEA is suspected, imaging the entire spine (cervical, thoracic, and lumbar) is highly recommended.

KEY FACT | **The classic triad of fever, back pain, and neurologic deficit is seen in less than 15% of patients at the time of initial presentation, and only 50% of this group are febrile initially.**

The goal of early diagnosis and treatment is to prevent further neurologic injury. The mortality rate is still estimated to be 10–23% despite surgery and antibiotics. The most important prognostic indicator of a patient's final neurologic outcome is the preoperative neurologic function. Patients who present with a neurologic deficit are less likely to regain full function, and those who have had neurologic symptoms longer than 36 hours are unlikely to regain any function.

KEY FACT | **The most important prognostic indicator of a patient's final neurologic outcome is the preoperative neurologic function.**

The fact that a patient is afebrile, has a normal neurologic examination, or has a normal WBC count should not dissuade the acute care clinician from performing a

more thorough evaluation with MRI if the pretest probability is moderate to high. Low-risk patients who are afebrile, have a normal exam, and have normal laboratory values can be discharged with close follow-up with their primary care clinician. If MRI and spine surgery capabilities are not immediately available, the acute care clinician should urgently transfer any high-risk patient to a center that has these resources.

Vertebral osteomyelitis

Pitfall | Starting antibiotics in patients in whom vertebral osteomyelitis is suspected but who have normal neurologic exams

The presentation of vertebral osteomyelitis can be similar to that of SEA. The risk factors and the causative organisms for osteomyelitis are the same as those for SEA. Elderly patients and those who have had recent surgery or urinary tract infections are at risk for infection with gram-negative organisms. Patients typically present with chronic back pain that has developed gradually over weeks and months. As in SEA, laboratory values (e.g. ESR, CRP, or WBC count) and plain radiographs cannot exclude the diagnosis and are not specific enough to confirm the diagnosis. MRI or CT is needed to confirm the diagnosis and will show abnormalities in 70–90% of patients with osteomyelitis [19].

Patients with osteomyelitis are treated with a 6–12-week course of antibiotics specific to the causative organism. The organism may need to be identified from bone biopsy or operative sample; therefore, it is recommended that antibiotics not be started until appropriate samples can be obtained or discussion is had with infectious disease specialists and spine surgeons. Antibiotics should not be withheld from patients who have a neurologic deficit on presentation.

The acute care clinician should obtain an MRI or CT scan emergently when the diagnosis is suspected and refer the patient to an appropriate spine surgeon if the diagnosis is confirmed. Short-term treatment consists of analgesics, with antibiotics being held until the case can be discussed with a spine surgeon.

KEY FACT | **In many cases the organism must be identified from bone biopsy or operative sample; therefore, it is recommended that antibiotics not be started until appropriate samples can be obtained.**

Spinal cord compression

Pitfall | Failure to consider spinal cord compression in patients who experience an increase in back pain when they are recumbent

Spinal cord compression (SCC) is a true medical emergency that has a multitude of causes, including herniated disks, cancer (primary or metastatic), epidural hematomas, epidural abscesses, and displaced vertebral fractures. The most common cause is a massive midline disk herniation, usually at the L4–L5 level [17]. Though the causes of SCC can vary, a consistent characteristic is that patients present with back pain that is worse with recumbency and ultimately associated with progressive neurologic deficits. Patients will go on to develop a bilateral or unilateral radiculopathy, followed by sensory deficits with symmetric and bilateral weakness. Saddle anesthesia with bowel, bladder, or erectile dysfunction is a late finding. Patients may also develop urinary incontinence due to overflow incontinency secondary to a neurogenic bladder.

KEY FACT | **Though the causes can vary, a consistent characteristic is that patients present with back pain that is worse with recumbency and ultimately associated with progressive neurologic deficits.**

As previously mentioned, the primary determinant of a patient's ultimate neurologic status is the neurologic status at the time of diagnosis. Therefore, it is imperative that the diagnosis be made early, prior to the development of any neurologic deficit. In fact, a normal neurologic examination should not exclude the diagnosis if the pretest probability of the presence of SCC is moderate to high. Unfortunately, laboratory studies cannot exclude the diagnosis. The diagnosis can be excluded only with MRI or, for those who cannot tolerate the MRI process, with CT myelography.

The acute care clinician should suspect SCC when patients complain of back pain that is worse when they are supine and is relieved when they sit up; if the patient has any neurologic deficits; if the patient has history of cancer or an HNP; or if risk factors for epidural hematoma (i.e. the patient is taking anticoagulants) or epidural abscess (see previous section) are present.

The ultimate treatment (e.g. surgery for a hematoma or abscess, or radiation for cancer) will be dictated by

the cause, but all patients can benefit from high-dose glucocorticoids.

Glucocorticoids minimize ongoing neurologic damage caused by compression by decreasing the edema in the spinal cord. Dosing varies and remains controversial, but dexamethasone 10 mg administered intravenously has been recommended. Higher-dose steroids are associated with more side effects (e.g. infection and hyperglycemia) but have better evidence for improving neurologic outcomes. There are no good randomized studies assessing neurologic outcomes following administration of low-dose glucocorticoids [20].

KEY FACT | **The ultimate treatment for spinal cord compression (e.g. surgery for a hematoma or abscess, or radiation for cancer) will be dictated by the cause, but all patients can benefit from high-dose glucocorticoids.**

The acute care clinician should start high-dose glucocorticoids and immediately transfer the patient to a center that has the capability to perform emergent MRI and that has a spine surgeon and radiation oncology services if they are not available at the initial location.

Abdominal aortic disease

Pitfall | Failure to consider the diagnosis of abdominal aortic aneurysm or aortic dissection in elderly patients with back pain

Low back pain is a significant complaint in more than 30% of adults 65 years or older. Although the majority of cases have a benign cause, one must consider vascular causes. Abdominal aortic aneurysm (AAA) and aortic dissection (AD) cause 1% of the deaths in this age group, accounting for 15,000 deaths annually in the United States [21]. Though the abrupt onset of severe back pain increases the suspicion for AAA rupture or an acute dissection, patients with these conditions can experience a chronic onset of back pain. In one case series, patients with unruptured AAA experienced back pain from 1 to 60 days (mean 11.6 days) before the diagnosis was made [22]. Making the correct diagnosis of AAA is further complicated by the fact that a pulsatile abdominal mass was noted in only 26% of patients who were misdiagnosed on their initial evaluation [23]. Other investigators have reported that the sensitivity of abdominal palpation in detecting a pulsatile mass is poor, reaching 80% only for aneurysms at least 5 cm in diameter [24–26].

KEY FACT | **Making the correct diagnosis of abdominal aortic aneurysm is further complicated by the fact that a pulsatile abdominal mass was noted in only 26% of patients who were misdiagnosed on their initial evaluation.**

Isolated back pain is a relatively rare presentation of a ruptured AAA. Lloyd *et al.* reported that only 4% of patients had back pain as their sole chief complaint, whereas 14% of patients had both abdominal and back pain [27]. In the series described by Yoshioka *et al.*, 77.5% of patients with AD had at least some back pain and 45% presented with sudden severe isolated back pain [28].

Clearly, not all elderly patients who present with low back pain need an evaluation for AAA or AD. However, the diagnosis of AAA or AD should be assumed in any patient who has back pain and syncope and should be strongly considered in hypertensive elderly patients with nontraumatic back pain of new onset. If the acute care clinician is facile with point-of-care ultrasound, an AAA study can be considered on anyone over the age of 50 who presents with abdominal, flank, or back pain [29].

Cancer

Pitfall | Failure to consider a primary cancer or metastasis to the spine as a cause of back pain

Cancer can metastasize to the spinal column and cause back pain. Pain can result from cancer growth within the epidural space (causing SCC), intramedullary growth within the vertebrae, or direct extension of the primary cancer into the vertebrae and spinal cord. Back pain is often the initial symptom of spinal metastasis and is often described as a constant dull pain that is not relieved with rest; it may increase with recumbency. The neurologic signs and symptoms associated with SCC (see earlier) are not uncommon.

If the patient has no neurologic deficits the initial evaluation can consist of plain radiographs, which may show destructive bony lesions. If destructive lesions are present and the patient has no neurologic deficits, it is acceptable to schedule an MRI scan within 24 hours to more thoroughly evaluate the lesions. In all other cases MRI is the imaging modality of choice and should be done emergently. As for SCC, glucocorticoids are recommended to

reduce spinal cord edema in patients with neurologic deficits. Typically, MRI scans are limited to the area in which the patient is having pain. However, in patients with cancer an entire spine MRI scan should be obtained, since they are at risk for metastasis at multiple levels and some lesions might not be symptomatic on the initial presentation.

If MRI and oncology services are not available in the facility where the patient is seen initially, the acute care clinician should consider transferring patients with cancer and back pain to a center with those resources.

Spinal compression fractures

Osteoporosis in the elderly can lead to spinal compression fractures without any preceding trauma. These fractures are a major cause of chronic pain and disability, limiting a patient's ability to pursue their daily activities. It is important to consider malignancy as a cause of the fracture.

Plain radiographs of the back should be obtained to exclude spinal compression fractures in the evaluation of patients over 65 years of age who present with back pain. If a fracture is seen and cancer is suspected, the patient should also undergo high-resolution CT of the spine. High-resolution CT can distinguish between benign and malignant causes of vertebral compression fractures and is more readily available than MRI [30].

A simple compression fracture can be treated with analgesics (e.g. NSAIDs, acetaminophen, or narcotics) and supportive care. The acute care clinician can also consider starting calcitonin, which has been shown to decrease the pain associated with compression fractures without the typical side effects seen with NSAIDs and narcotics [31, 32]. In the past elderly patients were often referred for vertebroplasty; however, the VERTOS IV study that compared vertebroplasty versus sham procedure showed no difference in pain scores at 1 month or 12 months [21, 33].

KEY FACT | **Calcitonin has been shown to decrease the pain associated with compression fractures without the typical side effects seen with nonsteroidal anti-inflammatory drugs and narcotics.**

Spinal stenosis

Spinal stenosis is most commonly found in the elderly with degenerative arthritis. Patients will typically complain of bilateral lower back pain that is worse with walking, prolonged standing, or spinal extension and relieved when the patient sits, lies down, or bends forward at the waist. Patients often complain of burning back pain that radiates to the buttocks and posterolateral legs when they are active. The symptoms are caused by stenosis of the vertebral canal, resulting from a combination of loss of intervertebral disk height, facet joint hypertrophy, and thickening of the ligament flavum. These changes cause a narrowing of the cross-sectional diameter of the spinal canal and neural foramina when the back is extended (i.e. when the patient is upright), which results in mechanical compression and nerve root ischemia.

Spinal stenosis can be diagnosed based on the history and physical examination alone. In the absence of any red flags there is no need for any diagnostic testing to be done in the acute setting. Patients should be treated with NSAIDs or acetaminophen and can complete their evaluation on a nonurgent basis. MRI is the study of choice, though CT myelography can be performed in those who are claustrophobic or have a contraindication to an MRI scan (e.g. a pacemaker). Patients with a neurologic deficit should undergo MRI or CT myelography immediately.

Pregnancy-related low back pain

Back pain is experienced by 50–90% of women during pregnancy [34]. The risk factors for back pain during pregnancy are advanced maternal age, a history of back pain, and the number of pregnancies. Neither fetal weight nor the amount of weight gained by the mother is associated with an increased risk of back pain [35]. The causes of back pain during pregnancy are multifactorial. Pregnant women are at risk for the development of spondylolysis and progression of their underlying spondylolisthesis. In fact, parous woman have a higher incidence of L4–L5 spondylolisthesis than their nulliparous counterparts [36]. Ectopic pregnancy can be considered in patients without a confirmed intrauterine pregnancy.

Pitfall | **Treating pregnancy-related back pain with nonsteroidal anti-inflammatory drugs**

The majority of patients can be treated successfully with a support binder or acetaminophen. NSAIDs should be avoided in pregnancy because of the risk of premature closure of the patent ductus arteriosus, preterm birth, and fetal renal abnormalities.

KEY FACT | **Back pain is experienced by 50–90% of women during pregnancy.**

In patients whose pain is not relieved with conservative measures, imaging studies may be required. Plain radiographs and CT are generally avoided as they expose the fetus to ionizing radiation. The fetus cannot be effectively shielded when obtaining lumbar spine radiographs. An MRI scan can be obtained and is completely safe in pregnancy; however, unless the mother is exhibiting neurologic deficits, it is unlikely that any findings will be acted on until after the baby is delivered. Therefore, reassurance, frequent examinations to confirm the absence of neurologic deficits, and pain control might be all that is needed to allow the patient to complete her pregnancy. If the pain does not resolve in the postpartum period, further diagnostic testing should be considered.

Pearls for improving patient outcomes

- All patients with back pain must be assessed for the presence of red flags that are markers of a serious underlying disease.
- Refer patients with red flags in their history or on physical examination for a more comprehensive evaluation, which may include laboratory testing, radiographs, computed tomography, or magnetic resonance imaging.
- Do not assume that a herniated nucleus pulposus is the cause of an individual's back pain.
- Use acetaminophen and nonsteroidal anti-inflammatory drugs as first-line treatment for pain control in patients with back pain. Prescribe narcotics and muscle relaxants only when necessary and for the shortest duration possible.
- Exercise is one of the few treatment modalities to be proven effective in the treatment of back pain.
- Consider the diagnosis of spinal epidural abscess in patients with fever, neurologic deficits, or risk factors for spinal epidural abscess when they present with atraumatic back pain.
- Start high-dose glucocorticoids as soon as possible in patients with spinal cord compression.
- Consider aortic dissection and aortic aneurysm in hypertensive patients who present with sudden onset of atraumatic back pain.
- Consider starting calcitonin in patients with vertebral compression fractures.

References

1 Wu A, March L, Zheng X, *et al*. Global low back pain prevalence and years lived with disability from 1990 to 2017: estimates from the global burden of disease study 2017. *Ann Transl Med* 2020; **8**(6): 299.

2 Haldeman S, Dagenais S. A supermarket approach to the evidence-informed management of chronic low back pain. *Spine J* 2008; **8**(1): 1–7.

3 Dagenais S, Caro J, Haldeman S. A systematic review of low back pain cost of illness studies in the United States and internationally. *Spine J* 2008; **8**(1): 8–20.

4 Dieleman JL, Baral R, Birger M, *et al*. US spending on personal health care and public health, 1996–2013. *JAMA* 2016; **316**(24): 2627–2646.

5 Leboeuf-Yde C, Kyvik KO. At what age does low back pain become a common problem? A study of 29,424 individuals aged 12–41 years. *Spine* 1998; **23**(2): 228–234.

6 Davis PJ, Williams HJ. The investigation and management of back pain in children. *Arch Dis Child Educ Pract Ed* 2008; **93**(3): 73–83.

7 Jensen MC, Brant-Zawadzki MN, Obuchowski N, *et al*. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med* 1994; **331**(2): 69–73.

8 Hayden JA, Ellis J, Ogilvie R, *et al*. Exercise therapy for chronic low back pain. *Cochrane Database Syst Rev* 2021; **9**(9): CD009790.

9 Chou R, Huffman LH, American Pain Society, *et al*. Medications for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Ann Intern Med* 2007; **147**(7): 505–514.

10 Chou R, Qaseem A, Snow V, *et al*. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. *Ann Intern Med* 2007; **147**(7): 478–491.

11 Jones CMP, Day RO, Koes BW, *et al*. Opioid analgesia for acute low back pain and neck pain (the OPAL trial): a randomised placebo-controlled trial. *Lancet* 2023; **402**(10398): 304–312.

12 Friedman BW, Chilstrom M, Bijur PE, *et al*. Diagnostic testing and treatment of low back pain in United States emergency departments: a national perspective. *Spine Phila Pa 1976* 2010; **35**(24): E1406–E1411.

13 Gottlieb M, Fagan MW, Polich ED. What is the role of systemic corticosteroids for radicular and nonradicular low back pain? *Ann Emerg Med* 2023; **82**(2): 164–166.

14 Maher CG. Effective physical treatment for chronic low back pain. *Orthop Clin North Am* 2004; **35**(1): 57–64.

15 Kim HS, Strickland KJ, Seitz AL, *et al*. Patient perspectives on seeking emergency care for acute low back pain and access to physical therapy in the emergency department. *Ann Emerg Med* 2023; **82**(2): 154–163.

16 Darouiche RO. Spinal epidural abscess. *N Engl J Med* 2006; **355**(19): 2012–2020.
17 Corwell BN. The emergency department evaluation, management, and treatment of back pain. *Emerg Med Clin North Am* 2010; **28**(4): 811–839.
18 Tompkins M, Panuncialman I, Lucas P, *et al*. Spinal epidural abscess. *J Emerg Med* 2010; **39**(3): 384–390.
19 Beronius M, Bergman B, Andersson R. Vertebral osteomyelitis in Goteborg, Sweden: a retrospective study of patients during 1990-95. *Scand J Infect Dis* 2001; **33**(7): 527–532.
20 Loblaw DA, Laperriere NJ. Emergency treatment of malignant extradural spinal cord compression: an evidence-based guideline. *J Clin Oncol* 1998; **16**(4): 1613–1624.
21 Broder J, Snarski JT. Back pain in the elderly. *Clin Geriatr Med* 2007; **23**(2): 271–289. v.
22 Cambria RA, Gloviczki P, Stanson AW, *et al*. Symptomatic, nonruptured abdominal aortic aneurysms: are emergent operations necessary? *Ann Vasc Surg* 1994; **8**(2): 121–126.
23 Marston WA, Ahlquist R, Johnson G Jr, *et al*. Misdiagnosis of ruptured abdominal aortic aneurysms. *J Vasc Surg* 1992; **16**(1): 17–22.
24 Fink HA, Lederle FA, Roth CS, *et al*. The accuracy of physical examination to detect abdominal aortic aneurysm. *Arch Intern Med* 2000; **160**(6): 833–836.
25 Lederle FA, Simel DL. The rational clinical examination. Does this patient have abdominal aortic aneurysm? *JAMA* 1999; **281**(1): 77–82.
26 Venkatasubramaniam AK, Mehta T, Chetter IC, *et al*. The value of abdominal examination in the diagnosis of abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 2004; **27**(1): 56–60.
27 Lloyd GM, Bown MJ, Norwood MG, *et al*. Feasibility of preoperative computer tomography in patients with ruptured abdominal aortic aneurysm: a time-to-death study in patients without operation. *J Vasc Surg* 2004; **39**(4): 788–791.
28 Yoshioka K, Toribatake Y, Kawahara N, *et al*. Acute aortic dissection or ruptured aortic aneurysm associated with back pain and paraplegia. *Orthopedics* 2008; **31**(7): 651.
29 Reardon R, Clinton M, Madore F, *et al*. Abdominal aortic aneurysm. In: Ma OJ, Matters JR (eds.), *Ma and Mateer's Emergency Ultrasound* (3rd edn.). New York: McGraw Hill Education Medical; 2014: pp. 225–245.
30 Kubota T, Yamada K, Ito H, *et al*. High-resolution imaging of the spine using multidetector-row computed tomography: differentiation between benign and malignant vertebral compression fractures. *J Comput Assist Tomogr* 2005; **29**(5): 712–719.
31 Lyritis GP, Ioannidis GV, Karachalios T, *et al*. Analgesic effect of salmon calcitonin suppositories in patients with acute pain due to recent osteoporotic vertebral crush fractures: a prospective double-blind, randomized, placebo-controlled clinical study. *Clin J Pain* 1999; **15**(4): 284–289.
32 Boucher E, Rosgen B, Lang E. Efficacy of calcitonin for treating acute pain associated with osteoporotic vertebral compression fracture: an updated systematic review. *CJEM* 2020; **22**(3): 359–367.
33 Firanescu CE, de Vries J, Lodder P, *et al*. Vertebroplasty versus sham procedure for painful acute osteoporotic vertebral compression fractures (VERTOS IV): randomised sham controlled clinical trial. *BMJ* 2018; **361**: k1551.
34 Anon. Impact of physical activity during pregnancy and postpartum on chronic disease risk. *Med Sci Sports Exerc* 2006; **38**(5): 989–1006.
35 Meleger AL, Krivickas LS. Neck and back pain: musculoskeletal disorders. *Neurol Clin* 2007; **25**(2): 419–438.
36 Sanderson PL, Fraser RD. The influence of pregnancy on the development of degenerative spondylolisthesis. *J Bone Joint Surg (Br)* 1996; **78**(6): 951–954.

CHAPTER 11
Pediatric Pitfalls

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Introduction

Caring for children in the acute care setting poses many challenges, with contributors including their physiologic differences from adults, early language development, and inherent apprehension of providers. These challenges lead the practitioner to either (1) overdiagnose certain common conditions such as acute otitis media as an explanation for the presenting complaint of fussiness or fever; or (2) substitute shotgun testing for a careful systematic history and physical examination. Both approaches are pitfalls in and of themselves, as more serious diagnoses can be overlooked or an unnecessary burden of diagnostic testing may be placed on a patient who is typically quite intolerant of anything invasive. In this chapter we review common pitfalls encountered by practitioners evaluating children in the acute care setting and provide clear guidance on how to avoid them.

Pitfall | **Not addressing the parental fear around fever in children**

Parents have many misconceptions and fears about fever, and they should be informed that it is not the height of the fever that is important but how the child looks (toxicity) and any associated symptoms such as dehydration, lethargy, or seizure.

Frequently parents are concerned that the temperature did not decrease after acetaminophen or ibuprofen. If a fever does not "break" with antipyretics, that does not mean that the infection is "more serious." In general, appropriately dosed acetaminophen or ibuprofen will bring the temperature down 1–1.5 °C (2–3 °F). High fevers (>40 °C, >104 °F) due to infectious causes do not cause brain damage. Body temperatures greater than 42.2 °C (108 °F) can cause brain damage, but those temperatures can only be reached from exogenous heat sources such as being left in an enclosed hot car.

Occasionally parents may notice some perioral cyanosis or paleness prior to the evolution of fever. This is due to arterial vasoconstriction that shunts warm blood to the core, making the venous blood more visible. The body is attempting to match the new higher hypothalamic set point due to pyrogens and interleukins.

Fever is not inherently harmful; it is the body's attempt to impair replication of microbes and may aid in phagocytic bactericidal activity. Fevers are treated to ease the child's discomfort. If the child is more comfortable, they will likely drink more fluids and stay hydrated through the illness. Once you have the child's weight, weight-based dosing of acetaminophen and ibuprofen should be provided to the caregivers rather than using age- or weight range-based dosing. Acetaminophen is dosed at 15 mg/kg every four hours and ibuprofen is dosed at 10 mg/kg every six hours.

Pitfall | **Failure to obtain a rectal temperature in the infant**

Fever is defined as a temperature of 38.0 °C (100.4 °F) or greater. Axillary and tympanic temperature measurements in infants and young children are unreliable [1]. The one exception for not taking a rectal temperature is

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when a child is potentially neutropenic from chemotherapy or other conditions, due to the possible risk of inducing bacteremia. Decisions on the appropriate workup of the infant are often based on an accurate temperature measurement (e.g. to trigger a neonatal sepsis evaluation). The temperature for children under two years of age should be taken rectally if possible.

Bundling of a young infant may increase the skin temperature but it does not increase the core temperature [2]. If you think the elevation is due to bundling, unbundle the child for 30 minutes and recheck the temperature. The parental assessment of fever by touch is likely to be accurate but should be confirmed by a thermometer prior to further evaluations [3]. This is particularly important in the neonate. Infants can have a rectal temperature as low as 36.2 °C (97.2 °F) or as high as 37.9 °C (100.2 °F), which is within the "normal" range. Like adults, children and infants have diurnal variation of their temperature, which has a peak between 5 and 7 p.m. In general, while parental reports of tactile fevers should be taken seriously, workup in the acute care setting should be based on the measured temperature.

Pitfall | **Not thinking systematically about a child with a fever**

The first step in fever management is determining that the child truly has a fever (a temperature >38 °C) and the second step is to risk stratify the child by age. Importantly, if the child is under 60 days of age, a neonatal sepsis pathway would be utilized. The third step applies to all ages: If the child looks truly ill or "toxic," then a complete sepsis evaluation should be performed and antibiotics considered. The fourth step is a good physical examination to look for any identifiable sources: hand foot mouth disease, bronchiolitis, strep pharyngitis, croup. In the absence of an identifiable source for the fever, assess the child's immunization status. If not fully immunized, the child is at increased risk of invasive bacterial infections and may warrant blood workup, especially for children under three years of age. If the child is fully immunized, consideration should be given to testing for urinary tract infection (UTI) and occult pneumonia. Past five days of true fever concern for Kawasaki disease (KD) or multisystem inflammatory syndrome in children (MIS-C) comes into play. These steps will be discussed in detail in this chapter.

Febrile infants

Pitfall | **Failure to follow the most current febrile infant guidelines**

Whether a febrile infant is managed in an urgent care setting or referred to a higher level of care, it is critical that providers give anticipatory guidance to parents. Knowledge of these guidelines will help providers effectively communicate anticipated plans so that the concerned parents understand the next steps. The most recent guideline for the management of the "well-appearing" febrile neonate was published by the American Academy of Pediatrics (AAP) in 2021 and incorporates data from four large febrile infant studies [4, 5]. The guideline separates infants into three discrete categories by age: age <22 days, 22–28 days, and >28 days. The reasoning behind the differences is that the rate of bacteremia varies by age [6]. The rate of bacteremia steadily decreases per week of life: 5.3% at two weeks, 3.3% at three weeks, and 1.6% by the fourth week. The guideline also emphasizes detecting *invasive* bacterial infections such as bacteremia and meningitis compared to the old terminology of *serious* bacterial infections, which included UTI.

There are two major studies (PECARN—Pediatric Emergency Care Applied Research Network; and Step-by-Step) and one overarching guideline that can help navigate the neonatal fever workup (Box 11.1) [7, 8]. Both studies and the guideline emphasize that the child needs to be well appearing without an identifiable source, such as respiratory syncytial virus (RSV) bronchiolitis.

Infant <22 days

The guideline and studies agree that the <22-day-old infant has a higher rate of invasive infection and a full sepsis evaluation (blood, urine, and cerebral spinal fluid [CSF] culture) and antibiotics are warranted. One debate in the <22-day-old is whether to test and treat for herpes simplex virus. The risk of acyclovir side effects is very low, while the benefit of preventing a devastating disease is high, so most practitioners treat the <22-day-old with acyclovir in addition to antibiotics, usually ampicillin and ceftazidime.

Infant 22–28 days

In evaluating the 22–28-day-old infant, the first step is to obtain a complete blood count (CBC) with differential, blood culture, urinalysis, urine culture, and inflammatory markers. Inflammatory markers can be a

Box 11.1 Low-risk criteria for well-appearing febrile infants

Previously healthy term infants without perinatal complications who are <60 days old and who are not found to have soft tissue, ear, or skeletal infection:

- Nontoxic appearance
- No previous use of antimicrobials
- Lack of a focus of infection on examination
- Over 22 days old

Low-risk laboratory criteria for well-appearing febrile infants:

- Blood:
  - Absolute neutrophil count <4000 or 5200/mm^3
  - Procalcitonin <0.5 ng/ml
  - C-reactive protein <20 mg/l
- Urine:
  - Gram stain negative, or
  - Leukocyte esterase and nitrate negative, or
  - Fewer than 5 WBC/hpf
- Cerebral spinal fluid (if performed):
  - Fewer than 8 WBC/mm^3
- Chest x-ray (if performed):
  - No infiltrate
- Stool (if diarrhea present):
  - Less than 5 WBC/hpf
  - No blood

hpf, high-power field; WBC, white blood cells.

Source: Pantell *et al*. [5], Kuppermann *et al*. [7], Gomez *et al*. [8].

C-reactive protein (CRP) or a procalcitonin, depending on the institution and what will give a timely result. A procalcitonin is considered more sensitive and specific for invasive bacterial infection than CRP or absolute neutrophil count (ANC) [9]. Abnormal inflammatory markers defined by the guideline are temperature >38.5 °C, procalcitonin >0.5 ng/ml, CRP >20 mg/l, and ANC > 4200 or 5200/mm^3.

Even with a positive urinalysis and normal inflammatory markers, most practitioners would continue with a full sepsis evaluation. In this age group the rate of bacteremia in the setting of a UTI is estimated at 20% [5]. While the AAP guideline does not give a specific recommendation regarding this scenario, both the PECARN and Step-by-Step infant studies suggest that a positive urinalysis would still place the infant in the high-risk category and still recommend a lumbar puncture, antibiotics, and hospital admission.

If CRP or procalcitonin is elevated, a full sepsis evaluation should be performed. If lumbar puncture will be delayed, antibiotics should be administered pending patient transfer to a higher level of care. If CSF is obtained, it is interpretable, and cell counts do not suggest central nervous system (CNS) infection, two reasonable options exist: observation at home with follow-up in 24 hours or hospital admission. For children sent home, a dose of ceftriaxone is recommended. If the child is observed in the hospital, the guidelines states that antibiotics (ceftriaxone) *may be* administered; this is where communication with the inpatient team is necessary and shared decision making with the family needs to occur.

Infant 29–60 days

In the older infant 29–60 days of age with fever, obtain urinalysis, urine culture, CBC with differential, blood culture, and inflammatory markers (procalcitonin and CRP). If inflammatory markers are normal, no further workup is needed. The infant should be observed closely at home with follow-up in 24 hours. No antibiotics are needed in this scenario.

If no source is identified and the inflammatory markers are abnormal, providers should engage in shared decision making with the parents about the risks and benefits of performing a lumbar puncture and the use of antibiotics. The rates of bacterial meningitis are low, 0.5–0.9% from the PECARN study, and likely higher with elevated inflammatory markers. If antibiotics are administered all efforts should be made to obtain CSF prior to administration. The child can be observed at home with close follow-up or admitted to the hospital for observation.

If the catheterized urinalysis suggests a UTI and inflammatory markers are normal, no lumbar puncture is needed. Based on local susceptibilities, the patient may be treated with a first- or third-generation cephalosporin, along with close follow-up in 24 hours. Because the PECARN and Step-by-Step studies do not offer prescriptive guidance for these situations in this age group, it is important to engage in thoughtful shared decision

making with the parents to clearly outline the risks and benefits of various approaches.

KEY FACT | **Well-appearing over one month old may be discharged home without antibiotics if they fulfill a low-risk history and laboratory criteria, have reliable parents, and have next-day follow-up.**

In the 1–3-month-old febrile infant with a known viral pathogen such as RSV or influenza, the likelihood of concomitant serious bacterial infection is low. Having a known antigen-positive test for RSV reduces the risk for serious bacterial illness from 9.6 to 2.2% [10]. Similarly, a positive rapid influenza test lowers the risk of serious bacterial infection from 17 to 2.6% [11]. In all cases the concomitant serious bacterial infection was a UTI. A study performed by Ralston *et al*. found that in the RSV-positive infant there were no cases of meningitis in 11 studies and no cases of bacteremia in 8 studies [12]. Therefore in the febrile neonate <60 days old with RSV bronchiolitis a catheterized urine is recommended.

This age group may, however, be challenging to manage in an urgent care setting and a low threshold for referrals to an emergency department (ED) is appropriate.

Pitfall | **Failing to incorporate immunization status in the decision making around children with fever without a source**

Fever >39 °C (102.2 °F) in the unimmunized child is concerning when a focus for the fever cannot be found. If any child appears ill or "toxic," referral should be made to an ED for a full sepsis evaluation and intravenous (IV) antibiotics. Even in unimmunized children the conjugated pneumococcal vaccine has decreased the rate of serious streptococcal infections through herd immunity. Nonetheless, the well-appearing unimmunized child with fever without a source needs to be evaluated for a possible UTI, pneumonia, and occult bacteremia.

Immunization status does not change the criteria for screening for a UTI in young children with fever without a source. Catheterized urine should be obtained in females <2 years of age, circumcised boys <6 months of age, uncircumcised boys <1 year of age, and any child with a prior UTI.

Children <3 years of age are at the greatest risk for occult bacteremia in the unimmunized or underimmunized child. For the otherwise well-appearing underimmunized child, one should look at how many *Haemophilus influenza* type B (HiB) and pneumococcal vaccines they have had; if the child has had at least two they should have partial immunity, reducing but not mitigating their risk. Once they reach three doses their immune system should be competent. For the unimmunized and underimmunized child there are two options for further evaluation. One option is to draw a blood culture and administer a dose of ceftriaxone. This approach recognizes that frequently, particularly with meningococcemia and *Salmonella* bacteremia, the white blood cell (WBC) count has limited value in predicting illness. The other approach is to obtain a blood culture and CBC with differential: If the WBC count is >15,000/mm^3 then treat with ceftriaxone [13]; if the WBC count is >20,000/mm^3 a chest x-ray should be performed to evaluate for occult pneumonia. The child should be followed up the next day for reevaluation. The absence of a definitive recommendation recognizes that there is not a clear balance between risk mitigation and antibiotic stewardship based on imperfect risk stratification.

KEY FACT | **A blood culture should be performed in the unimmunized and underimmunized child with fever without a source.**

Pitfall | **Being overly aggressive in obtaining blood cultures on a fully immunized, well-appearing child with fever without a source**

Vaccination with the conjugated pneumococcal vaccine and the HiB vaccine has dramatically changed the evaluation of the child under three years of age with fever [14]. The American College of Emergency Physicians developed a clinical policy to screen for occult bacteremia to reduce the risk of subsequent serious bacterial illnesses (pneumonia, cellulitis, septic arthritis, osteomyelitis, meningitis, or sepsis) [15]. These guidelines applied to previously healthy, well-appearing children aged 3–36 months with fever >39 °C (102.2 °F) without an identifiable source of infection. Since the introduction of the conjugated pneumococcal vaccine in 2000, invasive pneumococcal disease has dramatically declined [14]. Once a child has had two conjugated pneumococcal vaccines, (generally by four months of age) the risk for pneumococcal bacteremia is so low that it is no longer clinically indicated to draw empiric labs and blood culture [7, 13]. The child who is not immunized or has had fewer than two conjugated pneumococcal vaccines is at higher risk of bacteremia, specifically pneumococcal bacteremia. Empiric blood cultures should

be considered if the child has a temperature >40 °C (>104 °F), petechiae (*Meningococcemia*), prolonged gastroenteritis (*Salmonella*), or contact with meningococcal disease. While aggressive workups may seemingly provide reassurance, when they are unnecessary the risk of false positives outweighs the benefits of identifying remotely occult disease. These false positives can lead to additional unnecessary workups, cost, and parental angst.

KEY FACT | **Well-appearing children (3–36 months) who are fully immunized, with fever >39 °C (102.2 °F) without an identifiable source, do not require automatic blood cultures and antibiotics.**

Pitfall | Failure to consider urinary and pulmonary sources of infection in the child with fever without a source

UTIs are the most common occult infections in children and can present with subtle or no signs or symptoms other than fever. Therefore, consideration should be given to a possible UTI in children presenting with fever >39 °C (102.2 °F) and no obvious source. Children at highest risk for UTI include infants <6 months of age, uncircumcised boys <1 year of age, females <2 years of age, and any child with a previous UTI [16]. Factors that increase the sensitivity of detecting a UTI in girls <2 years of age are fever >39 °C (102.2 °F), fever for >2 days, white race, and age <1 year [17]. Urine should be obtained by urethral catheterization in all children who are not toilet trained. Urine collected by the bag method is unreliable and may be contaminated, leading to unnecessary treatment and testing. Urine testing by dipstick alone may miss a UTI in young infants. Urine culture with sensitivities should be performed on all specimens to increase the accuracy of diagnosis and treatment.

Antibiotic treatment for UTI depends on local susceptibilities of *Escherichia coli*, the predominant bacteremia in UTIs. In the authors' practice cephalexin has become a first-line oral antibiotic for treatment due to increasing trimethoprim-sulfamethoxazole resistance. Cefdinir is frequently used when there is a concern for local resistance of *E. coli*.

Infants <60 days old with a UTI are at higher risk for invasive bacterial illnesses and should be referred for a possible sepsis evaluation. Infants between three months and one year with febrile UTI (>39 °C, 102.2 °F) are also at increased risk of bacteremia and should be considered for referral to an ED for possible blood work and IV antibiotics, particularly if they appear ill or are feeding poorly [18].

Every child under three years of age with a UTI should have follow-up with their primary care provider for a renal ultrasound and consideration of a voiding cystourethrogram (VCUG) to detect any congenital renal collecting system abnormalities.

KEY FACT | **Urine samples in children who are not toilet trained should be obtained by urethral catheterization.**

Pitfall | Overutilization of chest x-rays

In children with fever without any obvious source, occult pneumonia also needs to be considered. Occult pneumonia is defined as a chest x-ray consistent with pneumonia and no respiratory distress or lower-airway signs or symptoms. Frequently there will be no tachypnea; they might have a slightly lower pulse oximetry level in the mid 90s instead of 99–100% [13]. Past fever guidelines recommended obtaining a CBC on children under three years of age with fever >39 °C (102.2 °F). Children found to have leukocytosis >20,000/mm^3 routinely had a chest x-ray performed. With the implementation of the conjugated pneumococcal vaccine, the incidence of occult pneumonia has decreased from 25 to 5% in highly febrile children with leukocytosis and no signs of pneumonia. Children presenting with only one day of fever and no cough are at extremely low risk for occult pneumonia and should not have a chest x-ray performed. The likelihood of detecting pneumonia increases with the duration of the cough, worsening cough, duration of the fever, and, if blood work is obtained, leukocytosis >20,000/mm^3 [19]. Usually after five days of persistent fever one should consider performing a chest x-ray to evaluate for occult pneumonia and start to consider KD and MIS-C.

KEY FACT | **Chest X-rays to evaluate for occult pneumonia should only be considered if the child has had fever for more than five days.**

Pitfall | Failing to consider Kawasaki disease or multisystem inflammatory syndrome in children (MIS-C) in a child with several days of fever

KD is a vasculitis of the medium-sized vessels that was formally known as mucocutaneous lymph node

syndrome [20]. The diagnosis requires at least five days of fever without other explanation and it typically occurs in children under five years of age. The signs associated with KD include conjunctivitis, redness of the lips or pharynx, puffy hands/feet or extremity changes, rash, and cervical lymphadenopathy. The conjunctivitis that is characteristic for KD is limbal sparing (while the sclera is red and injected, the area just around the iris remains white). Typically the child with KD is very fussy, uncomfortable, and sometimes irritable. Signs of KD may not appear simultaneously, so a good history is key to diagnosis. Undiagnosed KD can lead to the development of coronary aneurysms and cardiac involvement.

MIS-C is a syndrome that has emerged since the COVID-19 pandemic that reflects an exaggerated inflammatory response [21]. It occurs most frequently two to six weeks after a COVID-19 infection and typically affects older children and adolescents. Fever usually lasts at least four days without other known sources of infection. Considerable overlap exists with KD, with symptoms including gastrointestinal issues, rash, conjunctivitis, mucous membrane involvement, headache or lethargy, and even shock. Cough is an uncommon finding in the illness.

If a child has prolonged fever and there is clinical concern for KD or MIS-C, referral to the ED or admission is warranted. In addition to supportive care, these patients require an echocardiogram and may benefit from receiving IV immunoglobulin, aspirin, and steroids.

Pitfall | **Not treating the pain that accompanies an acute ear infection**

Acute otitis media (AOM) is the most frequent illness for which children may be placed on antibiotics. The diagnosis of AOM is based on three factors: color of the tympanic membrane (moderate to severe erythema); opacification or decreased translucency; and evidence of a middle ear effusion (moderate to severe bulging and/or no mobility on pneumatic otoscopy). AOM can be a self-limited illness that does not always require antibiotics, particularly in nonsevere cases as many are viral in nature. It has been shown that overall 60% of children with AOM will have resolution of symptoms in one day, whether treated with antibiotics or placebo [22]. Nonsevere AOM is defined as unilateral in nature, pain duration <48 hours, without "severe" pain, fever less than 39 °C (102.2 °F), and age over six months. In children with nonsevere AOM a 48–72-hour period of observation and analgesics are recommended before consideration of initiating an antibiotic. A wait-and-see prescription can be given to the family or the family can follow up in 48 hours. Parents are instructed to fill the prescription if there is worsening pain, fever >39 °C (102.2 °F), or ear drainage. For children with severe AOM who are under six months old with pain longer than two days, severe pain, or fever >39 °C (102.2 °F), an immediate prescription for antibiotics is given.

To address the pain associated with AOM, ibuprofen (10 mg/kg every six hours) or acetaminophen (15 mg/kg every four hours) should be given regularly for the first day after diagnosis. Anesthetic eardrops are also effective at reducing pain. Antipyrine benzocaine combination otic drops, five drops every three to four hours to the affected ear, have been shown to reduce ear pain compared to placebo [23]. Otic lidocaine can also be utilized but usually needs to be compounded at a local pharmacy [24]; 2% mucosal lidocaine (three to five drops every three hours) is a viable alternative. Anesthetic ear drops should not be used if there is any perforation of the tympanic membrane.

Once the decision to start antibiotics is made, we strongly recommend following guidance based on current local organism and prevalence patterns. Our current algorithm involves using high-dose amoxicillin (80–90 mg/kg divided twice daily) as first-line therapy. Therapy should be for 10 days in children under six years of age, and 5–7 days in children six years and older [25]. If the child has had amoxicillin within the last few months, we use amoxicillin-clavulanic acid (80–90 mg/kg divided twice daily) as second line followed by cefdinir (14 mg/kg once daily) as our third-line choice for more complicated patients.

> KEY FACT | **Over 60% of children with acute otitis media will have resolution of their pain in one day whether treated with antibiotics or placebo.**

Pitfall | **Not assessing for oral thrush when an infant has diaper candidiasis**

Infants acquire *Candida albicans* through maternal birth canal exposure, skin contact, and poor bottle sterilization. Over 20% of infants are colonized by the first month of life. Oral thrush in infants may produce pain

and consequently poor feeding. On physical examination, oral thrush appears as a white plaque over the inner cheek or tongue. Unlike milk, thrush typically cannot be scraped off with a tongue depressor. *Candida*, once present, colonizes the infant's entire gastrointestinal tract. When breakdown of the skin in the diaper region occurs, local infection can take hold. Over 30% of children with candidal diaper dermatitis will have oral thrush simultaneously. Candidal diaper dermatitis starts with redness of the anal mucosa and perianal skin, then spreads over the perineum and in severe cases to the thighs and abdomen. The eruption evolves into scaly papules that coalesce. The distinguishing feature of candidal diaper dermatitis is the presence of *satellite* papules. The satellite lesions are best described as pink to beefy red papules that spread from the intertriginous area of the diaper toward the legs or abdomen.

Treatment for oral thrush is nystatin oral suspension (100,000 units four times daily) for one to two weeks. Early in therapy all bottle nipples and pacifiers should be sterilized frequently in boiling water. Topical therapy of candidal diaper dermatitis includes nystatin, miconazole, or clotrimazole. It is important for breastfeeding mothers to apply the antifungal cream to their nipples to decrease reinfection of the infant.

Irritative diaper dermatitis is a very common skin disorder of infancy. Frequently this is due to impairment of the barrier function of the skin: moisture, friction, feces, and microorganisms all play a role. Primary irritative diaper dermatitis usually starts when skin is exposed to moisture for a prolonged period of time. Chemical irritation from urine and feces increases the penetration of bacteria and fungi, resulting in severe inflammation. The rash will typically start pink and progress to beefy red as it becomes more severe. Areas of friction and irritation from the diaper are affected most. Typically this will be over the perineum and buttocks, sparing the gluteal cleft and the leg folds. In the areas of greatest irritation, scaling or ulceration may be seen. *C. albicans* should be considered part of the pathologic process if the irritative dermatitis has been present for more than three days or if the child has recently been on antibiotics [26].

First-line therapies for diaper rash include increased frequency of diaper changes, superabsorbent disposable diapers, applying water-repellant barrier creams (zinc oxide), adding bath oil to bath water, and bathing the baby once or even twice a day. Second-line treatment includes treating with an antifungal ointment such as nystatin or applying 1% hydrocortisone ointment. Interestingly, the topical antibacterial agent mupirocin applied four times daily works as an antifungal agent and is effective in diaper dermatitis [27]. Other steps include avoidance of irritative diaper wipes that contain fragrance, benzalkonium chloride, isothiazolinone, and alcohol. Warm water, mild soap, and cotton balls are mild and effective for cleaning the diaper area.

KEY FACT | **The first steps to treat diaper dermatitis are to decrease the moisture in the diaper by frequent changes and to increase the barrier between that moisture and the child with water-repellant barrier creams. Children with simultaneous oral and diaper candidiasis should be treated with oral and topical nystatin.**

Pitfall | Not considering foreign-body aspiration in a young child with wheezing

Foreign-body aspiration (FBA) is a potentially life-threatening emergency that has a range of presentations. FBAs are frequently misdiagnosed initially as asthma, pneumonia, a choking spell, or upper respiratory infection [28]. Many children with FBA will be between the ages of one and three years and have a sudden onset of coughing or choking. Food items, especially peanuts, are the most common culprits for aspiration. Other common objects aspirated include other nuts and seeds, apples, carrots, hot dogs, and popcorn. On examination the child might have a cough, wheeze, or dyspnea. Unlike adults, in young children the bifurcations of the mainstem bronchi are at the same angle, making the left bronchus equally open to FBA as the right. Asymmetric breath sounds are heard in the minority of children with FBA.

Factors that contribute to delayed diagnosis are lack of a witnessed choking spell and normal radiographs on initial evaluation. Initial radiographs were normal in over 30% of children diagnosed with FBA by bronchoscopy. One must maintain a high index of suspicion for FBA in young children, especially in a young child with a choking spell, first-time wheezing, or persistent cough. Inspiratory and forced expiratory or bilateral decubitus chest radiographs may be obtained in stable cases to evaluate for evidence of air trapping. A normal chest radiograph does not exclude an FBA; if there is reasonable concern, the child should be referred for further evaluation and possible bronchoscopy.

KEY FACT | **The classic triad of wheeze, cough, and decreased breath sounds is present in only in one-third of cases of pediatric foreign-body aspiration.**

Pitfall | Not looking for the double-ring sign of a disc battery when a coin-shaped object is ingested

Of all foreign-body ingestions, 80% occur in young children (six months to three years of age) due to their curiosity and propensity to explore the world through their mouths. Unlike older children who are likely to report a history of accidental or intentional foreign-body ingestion, young children may have very nonspecific symptoms such as irritability, drooling, or food refusal, or they may even be asymptomatic. Coins are the most common ingested object and the vast majority of ingested objects are radiopaque. Management of the ingestion depends not only on the type, size, and shape of the object ingested but also the location of the object on detection.

Esophageal foreign bodies typically lodge at three distinct points along the esophagus: the level of the cricopharyngeus, at the thoracic inlet, or at the lower esophageal sphincter [29]. Up to 40% of patients with known esophageal coins may be completely asymptomatic. Radiographs of the chest (including a lateral of the chest and neck) and abdomen should be obtained. Esophageal coins will appear as a disc on the anteroposterior (AP) view and on edge on the lateral view. Extreme care must be taken to differentiate disc batteries from coins, as the former can rapidly damage the esophageal mucosa as soon as one hour after ingestion. Radiographs of button batteries will have a double density or rim, which is best detected on the lateral view.

When a button battery is identified in the esophagus, immediate removal should be undertaken. Oral administration of honey is recommended for any case where delay in removal is possible. Other circular objects lodged in the upper third of the esophagus should be removed within 12–16 hours, and obviously sooner if there is airway compromise or distress. Objects in the middle or lower third can be observed for up to 24 hours and will often spontaneously pass. Removal is recommended for impaction greater than 24 hours. An alternative to observation—which can be safely performed in the urgent care setting in selected patients without underlying esophageal abnormalities and known short duration of impaction—is the use of an esophageal dilator (bougienage) to force the object into the stomach. A 95.4% success rate was reported in a retrospective review of 372 patients undergoing bougienage treatment for esophageal coins [30]. This technique was associated with a significantly decreased length of stay and cost savings over hospitalization and endoscopy. Sharp objects lodged within the esophagus should be immediately removed, typically via rigid or flexible esophagoscopy, due to their high risk for perforation and low likelihood of spontaneous passage.

Once an object has entered the stomach, passage is directly related to the size and length of the object. Most round objects less than 1 cm × 3 cm will spontaneously pass. Coins in the stomach can be safely observed for two to four weeks for spontaneous passage as long as the patient remains asymptomatic. If the object is not identified in the stool within four weeks, repeat radiographs to confirm passage or retention are recommended. Endoscopic removal of a coin that has not passed out of the stomach by this time is generally recommended. Long, nonsharp objects may pass even if they are up to 5–6 cm in length as long as their diameter is <2 cm. Sharp objects in the stomach or proximal duodenum are often removed due to the higher risk of perforation (up to 35%), but once these objects have moved beyond the duodenal sweep observation is recommended unless signs of perforation such as fever or peritoneal signs develop. Consultation with a gastroenterologist for any identified or suspected ingested sharp object in the stomach, no matter what the size, is reasonable.

Magnets used in children's toys have become more common and present a unique problem when ingested. While a single smooth-edged magnet poses little to no risk, multiple magnets can attach to each other across loops of bowel, creating an obstruction. Radiographic evidence of more than one magnet in the stomach, unless these magnets are already attached to each other, should prompt immediate removal. Once magnets have traversed the pylorus, observation of signs of bowel obstruction or development of significant abdominal pain that could indicate perforation would guide surgical removal.

KEY FACT | **Some 90% of all ingested foreign bodies will pass spontaneously.**

Pitfall | **Failure to use a systematic approach to the evaluation of the fussy, well-appearing infant**

There are few diagnostic dilemmas more challenging than the well-appearing fussy or crying infant. Parents of these children often want answers as to why their child is crying excessively and may themselves be quite emotional. A provider can quickly become overwhelmed by these emotions, or the fear of missing a potentially devastating diagnosis, and embark on a rabbit trail of testing. Alternatively, there may be a tendency to prematurely label the child as having infantile colic before ruling out other possibilities. Colic is a diagnosis of exclusion, thus it is important to rule out a variety of other causes for the crying infant. An astute clinician armed with a good differential and a thorough physical examination can rule in or out most of the common causes for the well-appearing crying infant. Additional testing can be based on the presence or absence of other findings (Box 11.2).

Corneal abrasions are common yet quite difficult to detect without fluorescein staining. An infant will not necessarily present with unilateral tearing, but may have asymmetric redness of one eye and the crying is typically sudden in onset. Crying will often resolve after instillation of an ophthalmic anesthetic prior to placement of the fluorescein that confirms the diagnosis. Oral candidiasis can be a normal finding in infants and is sometimes asymptomatic. When associated with poor feeding, a trial of antifungals can be initiated. Coxsackie viral infection (herpangina) or primary herpes gingivostomatitis may have subtle initial presentations, so close examination of the oral cavity should not be overlooked. Otitis media is often overcalled as a cause of crying in an infant. The diagnosis should be certain, based on clinical findings including bulging tympanic membrane, erythema, and opacificity. Treatment of the pain with numbing ear drops and oral analgesics should produce resolution of the crying if otitis is indeed the cause.

Box 11.2 Some potential causes of fussiness in a well-appearing infant

Head, eyes, ears, nose, and throat (HEENT):
- Corneal abrasion; oral candidiasis; coxsackie viral herpangina; herpes gingivostomatitis; otitis media

Gastrointestinal/genitourinary:
- Constipation; intussusception; gastroesophageal reflux; rectal fissure; hernia; testicular torsion; urinary tract infection

Cutaneous:
- Hair tourniquet; skin or soft tissue abscess; diaper dermatitis: group A streptococcal perianal dermatitis; *Candida*; contact dermatitis

Miscellaneous/other:
- Recent immunizations; abuse; supraventricular tachycardia; infantile colic

Intermittent fussiness in a somewhat predictable or rhythmic pattern can be seen in infants with constipation. A careful history of stooling frequency and consistency along with a digital rectal examination will provide important clues in making this diagnosis. Close examination of the rectal mucosa for a rectal fissure is done at the same time. Intussusception can also produce similar symptoms to constipation. Infants may be completely normal between episodes and a high index of suspicion is required. Intussusception, however, is relatively rare under six months of age. When it is suspected, a focused colonic ultrasound should be performed.

Timing of the onset of crying can provide important clues. Infants who become very fussy shortly after eating with associated arching of the back and turning of their head (Sandifer syndrome) may suffer from gastroesophageal reflux. There usually is a clear association with feedings. A trial of an acid-blocking agent can help to confirm this diagnosis.

Certain routine immunizations are also associated with fussiness. Diphtheria–tetanus–acellular pertussis (DTaP), conjugated pneumococcal, and oral rotavirus vaccines all indicate fussiness or irritability as common side effects on the vaccine information sheets. DTaP has also been associated with nonstop crying for more than three hours in 1 out of 1000 infants administered this vaccine.

It is critical to completely undress the infant to search for hair or other fiber tourniquets, which can be found on any of the fingers or toes, penis, labia, or even clitoral regions. A thorough skin examination looking for bruising may indicate the possibility of abuse. Tearing of the frenulum or intraoral or face and neck bruising are very concerning findings in infants for abuse. A complete musculoskeletal examination may highlight an area of swelling or tenderness associated with a fracture or redness and swelling secondary to a septic joint. Attention to the diaper region is also important, looking for subcutaneous abscesses seen in community-acquired

methicillin-resistant *Staphylococcus aureus*. This organism has a predilection for the diaper region in infants due to rectal colonization. Severe diaper dermatitis caused by group A beta-hemolytic *Streptococcus*, *Candida* species, or even contact dermatitis can be painful and produce crying. Genitourinary examination looking for scrotal or labial swelling, if nonreducible, may indicate an incarcerated or strangulated inguinal hernia. An abnormal testicular lie with swelling suggests testicular torsion.

Supraventricular tachycardia in infants is often asymptomatic. However, if an infant is found to have an extreme tachycardia with no identifiable cause to suggest a compensatory sinus tachycardia, an electrocardiogram (ECG) looking for the absence of p-waves and a rhythm strip to identify the absence of variability should be obtained. Signs of heart failure are late phenomena in the otherwise healthy infant with no underlying structural heart abnormalities.

When no alternative source for the crying can be identified, in the infant under three months of age a UTI should be ruled out with a catheterized specimen, even in the absence of fever. Other routine laboratory or imaging testing is not recommended.

KEY FACT | **Thorough examination, not extensive testing, of the fussy or crying infant is essential in localizing a source for the discomfort.**

Pitfall | **Overutilization of neuroimaging of the minor head-injured child**

Traumatic brain injury is one of the leading causes of death and disability in children worldwide. In the United States alone there are an estimated 1.5–3.8 million head injuries in children each year. The majority of these injuries are minor, yet there remains a small but significant percentage of patients with relatively unimpressive presenting signs or symptoms who have suffered a clinically important injury.

Identification of the child who is at low risk for clinically important traumatic brain injury (ciTBI) is necessary for the acute care provider, both to identify important injuries and to minimize overtesting or referral. There is a great deal of parental and provider anxiety over missing these injuries, which has contributed to a dramatic rise in the use of computed tomography (CT) scanning. Concurrently there has also been a recognition of and growing concern over the potential risks of ionizing radiation to the developing brain. A robust clinical decision rule with a negative predictive value for ciTBI of 100% was prospectively derived, validated, and published to assist providers in the evaluation of this patient population (Table 11.1) [31]. The authors identified higher-risk factors and lower-risk factors for ciTBI and, based on these findings, described a tiered approach to patients. For example, a child over two years of age with only vomiting would not necessarily require neuroimaging, as the risk for ciTBI is <0.9%. In this case, the authors recommend a period of observation versus imaging, based on clinician experience. Utilization of this prediction rule can decrease imaging in very low-risk and low-risk patients after minor head injury through either reassurance or active observation by care providers for worrisome changes. Single versus combinations of risk factors lower or potentially raise suspicion for ciTBI. Although the tool was derived in patients presenting to the ED within 24 hours of injury, a subsequent study looked at delayed presentation and found the rule to be satisfactory [32]. Reason suggests that the rule could dramatically decrease the exposure of children to potentially harmful ionizing radiation or unnecessary referral for imaging.

Pitfall | **Failure to manage concussions according to guidelines**

A concussion is defined as a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces [33]. Clinically more important are some key features of concussions that assist in making the diagnosis. A concussion can be sustained either through a direct blow to the head or neck or through transmitted impulsive forces such as from a blow to the body or from rapid deceleration in a motor vehicle accident. Loss of consciousness is not a prerequisite to a diagnosis that is clinically based on the presence of symptoms in one or more of the following realms: physical, cognitive, emotional, or sleep related. Lists of commonly reported symptoms within these categories are widely available and symptoms of concussion typically fall into one of those four realms. While patients can complain of a host of different types of physical symptoms, the most reported ones are headache, nausea, vomiting, imbalance, visual problems, fatigue, sensitivity to light or sounds, and feeling "stunned." Cognitive symptoms may manifest as feeling mentally "foggy," sluggish or slow; concentration difficulties; confusion, forgetfulness,

Table 11.1 Decision rule for identifying children at low risk for clinically important traumatic brain injury (ciTBI).

Age <2 years		
GCS = 14 or other signs of altered mental status[a] or signs of depressed skull fracture	→	CT scan indicated (4.4% risk of ciTBI)
Nonfrontal scalp hematoma, or history of LOC ≥5 s, or severe mechanism of injury,[b] or not acting normally per parent	→	Consider observation vs. CT scan (0.9% risk of ciTBI)
None of the above risk factors	→	CT scan not indicated (<0.02% risk of ciTBI)
Age 2–18 years		
GCS = 14 or other signs of altered mental status[a] or signs of basilar skull fracture	→	CT scan indicated (4.3% risk of ciTBI)
History of LOC, or history of vomiting, or severe mechanism of injury,[b] or severe headache	→	Consider observation vs. CT scan (0.9% risk of ciTBI)
None of the above risk factors	→	CT scan not indicated (<0.05% risk of ciTBI)

CT, computed tomography; GCS, Glasgow Coma Scale; LOC, loss of consciousness.
[a] Agitation, somnolence, repetitive questioning, or slow to respond to verbal communication.
[b] Motor vehicle crash with patient ejection, death of another passenger, or rollover; pedestrian or bicyclist without a helmet struck by a motorized vehicle; falls of more than 1 m (3 ft) (age <2 years) or 1.5 m (5 ft) (age >2 years); or head struck by high-impact object.
Source: Adapted from Kuppermann *et al*. [31].

or repetitive questioning; and slowness while answering questions. Patients with concussion will often report the presence of emotional lability, sadness, nervousness, or even a flattened affect. Excessive sleepiness or drowsiness, and difficulty falling asleep or staying asleep, are some of the sleep-related disturbances associated with concussions.

It is important to note that neurologic dysfunction represents a functional and not a structural disturbance, and thus most patients with a concussion will have completely normal neuroimaging if obtained. Thus, a negative CT scan, or falling into the low-risk category based on a clinical decision, does not rule out the possibility of a patient still having a concussion.

When a patient meets the clinical diagnosis of a concussion, it is not necessary to attempt to assign a "grade" to the concussion. The currently accepted approach to determining the timing of return to activity is based on the evaluation of symptom resolution rather than concussion grading. Thus the clinician must be able to identify patients who have sustained a concussion and appropriately refer them for follow-up. For the more severely affected patients it may be necessary to impose both physical and cognitive restrictions to minimize symptoms and speed up recovery. The magnitude of these restrictions (e.g. complete versus partial physical and cognitive rest) should be recommended by the initial provider, but the duration of these restrictions is tailored to the individual and cannot be accurately predicted ahead of time. Close follow-up is essential.

Most concussive symptoms are short lived, with most patients experiencing complete resolution in 7–10 days. Patients with repeated concussions or delayed presentation to care, and a history of a mechanism consistent with a probable concussion and prolonged symptoms, may require referral to a specialist in dealing with traumatic brain injury. All other cases can be referred to the primary care provider.

KEY FACT | **Utilizing a simple clinical decision rule for minor head injury can significantly reduce the use of computed tomography scans in children presenting with minor head injury, but does not rule out the possibility of a concussion.**

Pearls for improving patient outcomes

- The clinical appearance of a young infant (<60 days old) presenting with fever is not reliable in determining whether a sepsis evaluation should be performed to evaluate for serious bacterial illnesses.
- Parents should be given weight-based dosing of ibuprofen or acetaminophen to treat pain or fever effectively.

• Most ear infections are viral in nature and only require adequate pain control with ibuprofen or acetaminophen and otic drops, not antibiotics.
• Fully immunized, well-appearing children (<36 months old) with high fever (>39 °C, 102.2 °F) for more than two days and no apparent source of infection on history or physical examination should have a urine test and/or a chest x-ray performed to evaluate for a possible occult bacterial infection.
• Consider inflammatory syndromes in children with prolonged fever and no other source.
• Diaper rash is the result of skin breakdown due to an overly moist diaper environment. To effectively treat it, a strong barrier cream, superabsorbent diapers, and frequent diaper changes should be used.
• Foreign-body aspiration should be considered in any young children with a history of a choking spell or sudden-onset coughing illness. Radiographs are frequently "normal." If the suspicion is high, referral should be made for bronchoscopy.
• With any coin lodged in the esophagus, always consider the possibility that it may be a disc battery. Look for the distinctive double ring that is seen with batteries.
• Colic is a diagnosis of exclusion; fussy infants require a thorough stepwise evaluation.
• Utilization of a simple clinical decision rule can help to classify patients with minor closed head injury into low-risk, very low-risk, and high-risk groups to guide imaging decisions.
• Concussion is a clinical diagnosis. Patients diagnosed with a concussion should have limitations in their physical and cognitive activity and should be fully recovered prior to a return to sporting activities.

References

1 Alayed Y, Kilani MA, Hommadi A, *et al*. Accuracy of the axillary temperature screening compared to core rectal temperature in infants. *Glob Pediatr Health* 2022; **9**: 2333794x221107481.
2 Grover G, Berkowitz CD, Lewis RJ, *et al*. The effects of bundling on infant temperature. *Pediatrics* 1994; **94**(5): 669–673.
3 Li YW, Zhou LS, Li X. Accuracy of tactile assessment of fever in children by caregivers: a systematic review and meta-analysis. *Indian Pediatr* 2017; **54**(3): 215–221.
4 Roberts KB, Pantell RH. Development of the new AAP febrile infant clinical practice guideline. *Hosp Pediatr* 2021; **11**(9): 1028–1032.
5 Pantell RH, Roberts KB, Adams WG, *et al*. Evaluation and Management of Well-Appearing Febrile Infants 8 to 60 days old. *Pediatrics* 2021; **148**(2): e2021052228.
6 Powell EC, Mahajan PV, Roosevelt G, *et al*. Epidemiology of bacteremia in febrile infants aged 60 days and younger. *Ann Emerg Med* 2018; **71**(2): 211–216.
7 Kuppermann N, Dayan PS, Levine DA, *et al*. A clinical prediction rule to identify febrile infants 60 days and younger at low risk for serious bacterial infections. *JAMA Pediatr* 2019; **173**(4): 342–351.
8 Gomez B, Mintegi S, Bressan S, *et al*. Validation of the "step-by-step" approach in the management of young febrile infants. *Pediatrics* 2016; **138**(2): e20154381.
9 Milcent K, Faesch S, Gras-Le Guen C, *et al*. Use of procalcitonin assays to predict serious bacterial infection in young febrile infants. *JAMA Pediatr* 2016; **170**(1): 62–69.
10 Bilavsky E, Shouval DS, Yarden-Bilavsky H, *et al*. A prospective study of the risk for serious bacterial infections in hospitalized febrile infants with or without bronchiolitis. *Pediatr Infect Dis J* 2008; **27**(3): 269–270.
11 Mintegi S, Garcia-Garcia JJ, Benito J, *et al*. Rapid influenza test in young febrile infants for the identification of low-risk patients. *Pediatr Infect Dis J* 2009; **28**(11): 1026–1028.
12 Ralston SL, Garber MD, Rice-Conboy E, *et al*. A multicenter collaborative to reduce unnecessary care in inpatient bronchiolitis. *Pediatrics* 2016; **137**(1). https://doi.org/10.1542/peds.2015-0851.
13 Shah S, Bachur R, Kim D, *et al*. Lack of predictive value of tachypnea in the diagnosis of pneumonia in children. *Pediatr Infect Dis J* 2010; **29**(5): 406–409.
14 Wilkinson M, Bulloch B, Smith M. Prevalence of occult bacteremia in children aged 3 to 36 months presenting to the emergency department with fever in the postpneumococcal conjugate vaccine era. *Acad Emerg Med* 2009; **16**(3): 220–225.
15 Baraff LJ, Bass JW, Fleisher GR, *et al*. Practice guideline for the management of infants and children 0 to 36 months of age with fever without source. Agency for Health Care Policy and Research. *Ann Emerg Med* 1993; **22**(7): 1198–1210.
16 Gorelick MH, Hoberman A, Kearney D, *et al*. Validation of a decision rule identifying febrile young girls at high risk for urinary tract infection. *Pediatr Emerg Care* 2003; **19**(3): 162–164.
17 Shaw KN, Bachur RG, Gorelick MH. Perspectives on urinary tract infection and race. *JAMA Pediatr* 2020; **174**(9): 910–911.
18 American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Pediatric Fever, Mace SE, Gemme SR, *et al*. Clinical policy for well-appearing infants and children younger than 2 years of age presenting to the emergency department with fever. *Ann Emerg Med* 2016; **67**(5): 625–39 e13.

19 Shah S, Mathews B, Neuman MI, *et al*. Detection of occult pneumonia in a pediatric emergency department. *Pediatr Emerg Care* 2010; **26**(9): 615–621.
20 Faye A. Kawasaki disease: a new understanding of the clinical spectrum. *Lancet Child Adolesc Health* 2023; **7**(10): 672–673.
21 Tiwari V, Daniel AA. Multisystem inflammatory syndrome in children: a year in review. *Eur J Rheumatol* 2022; **9**(3): 167–175.
22 Lieberthal AS, Carroll AE, Chonmaitree T, *et al*. The diagnosis and management of acute otitis media. *Pediatrics* 2013; **131**(3): e964–e999.
23 Hoberman A, Paradise JL, Reynolds EA, *et al*. Efficacy of Auralgan for treating ear pain in children with acute otitis media. *Arch Pediatr Adolesc Med* 1997; **151**(7): 675–678.
24 Bolt P, Barnett P, Babl FE, *et al*. Topical lignocaine for pain relief in acute otitis media: results of a double-blind placebo-controlled randomised trial. *Arch Dis Child* 2008; **93**(1): 40–44.
25 Hoberman A, Paradise JL, Cohen R. Duration of therapy for acute otitis media. *Pediatr Infect Dis J* 2000; **19**(5): 471–473.
26 Benitez Ojeda AB, Mendez MD. Diaper dermatitis. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2023. https://www.ncbi.nlm.nih.gov/books/NBK559067. Accessed July 23, 2024.
27 de Wet PM, Rode H, van Dyk A, *et al*. Perianal candidosis—a comparative study with mupirocin and nystatin. *Int J Dermatol* 1999; **38**(8): 618–622.
28 Lee JJW, Philteos J, Levin M, *et al*. Clinical prediction models for suspected pediatric foreign body aspiration: a systematic review and meta-analysis. *JAMA Otolaryngol Head Neck Surg* 2021; **147**(9): 787–796.
29 Gurevich Y, Sahn B, Weinstein T. Foreign body ingestion in pediatric patients. *Curr Opin Pediatr* 2018; **30**(5): 677–682.
30 Arms JL, Mackenberg-Mohn MD, Bowen MV, *et al*. Safety and efficacy of a protocol using bougienage or endoscopy for the management of coins acutely lodged in the esophagus: a large case series. *Ann Emerg Med* 2008; **51**(4): 367–372.
31 Kuppermann N, Holmes JF, Dayan PS. Head injury decision rules in children. *Lancet* 2017; **390**(10101): 1487–1488.
32 Borland ML, Dalziel SR, Phillips N, *et al*. Delayed presentations to emergency departments of children with head injury: a PREDICT study. *Ann Emerg Med* 2019; **74**(1): 1–10.
33 Kazl C, Torres A. Definition, classification, and epidemiology of concussion. *Semin Pediatr Neurol* 2019; **30**: 9–13.

CHAPTER 12
Geriatric Pitfalls

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Introduction

According to the US Census Bureau, the number of adults 65 years of age and older is anticipated to increase by 35 million, more than 60%, between 2022 and 2065. As such, older adults are projected to represent over a quarter of the US population, up from just 17% in 2022 [1]. A similar increase is anticipated globally, with a 53% increase in those over the age of 65 between 2022 and 2065 to an estimated 1.98 billion older adults [2].

Older adults pose unique challenges to acute care practitioners. Common diseases often present atypically in this patient population, laboratory values and vital signs may be more challenging to interpret, comorbid conditions may complicate straightforward medical issues, and social support systems may make it difficult for patients to handle seemingly innocuous problems such as sprained ankles.

Pitfall | **Misinterpretation of "normal" vital signs**

Most practitioners evaluate a patient's vital signs as the first step of any encounter. However, this straightforward practice may be challenging in the case of the geriatric patient. Both seemingly "normal" vital signs as well as "abnormal" vital signs, without appropriate considerations within the context of the geriatric patient, may lead the provider astray.

Older adults are often normothermic or even hypothermic despite infectious pathology [3]. Tachycardia that would normally be expected with serious conditions may be absent due to intrinsic conduction system abnormalities or may be blunted by medications such as beta-blockers. Literature has highlighted the importance of tachycardia in geriatric trauma patients where even mild tachycardia, 90–100 beats per minute, was associated with increased mortality [4].

Chronic hypertension is one of the most common diseases, with a prevalence of up to 80% in people over the age of 70 [5]. A "normal" blood pressure may represent significant hypoperfusion in a patient with underlying chronic hypertension. The trauma literature has shown that occult hypoperfusion in geriatric patients can be seen with systolic blood pressures (SBPs) in the range of 130–150 mmHg [6]. A study of nearly 25,000 trauma patients identified the optimal definition of hypotension after trauma as being an SBP of 120 mmHg in patients aged 50–69 years and 140 mmHg in patients 70 years and older [7]. Pain is often described as the "fifth vital sign." Even in this area, older adults may not be as reliable. Altered pain perception among this population is well documented. A study from the 1960s demonstrated a prolongation in time necessary to both sense a painful stimulus and then to perceive it as painful [8]. Endoscopically proven peptic ulcer disease (PUD) is painless only 8% of the time in young cohorts, but in patients above the age of 60 it may be seen in up to 35% of cases [9].

Given the frequency of hypertension in this patient population, it is also important to recognize that isolated hypertensive readings without end-organ dysfunction, sometimes referred to as "hypertensive

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urgency," do not require aggressive treatment or referral to the emergency department (ED). In the absence of end-organ dysfunction such as stroke, acute coronary syndrome (ACS), and aortic dissection, aggressive short-term treatment of systolic and diastolic blood pressures of at least 180 mmHg and 110 mmHg, respectively, does not show improved outcomes [10]. Patients with sustained elevated blood pressures discovered in the urgent care setting should be encouraged to seek primary care evaluation to begin longitudinal treatment for hypertension.

Pitfall | **Failure to appreciate atypical presentations of acute coronary syndrome**

KEY FACT | **The most common "atypical" symptoms of acute coronary syndrome (ACS) in the elderly are dyspnea, diaphoresis, nausea/vomiting, and syncope. Only 35% of patients over the age of 84 will have an ST-elevation myocardial infarction as their presentation of ACS.**

People over the age of 60 account for 65% of all cases of ACS and 80% of all deaths from ACS. ACS can present atypically in older adults. In the Global Registry of Acute Coronary Events (GRACE), 8.4% of patients with ACS presented without chest pain. In older adults, the absence of chest pain can be as high as 22% in those with ACS [11]. Of patients without chest pain, nearly one-quarter were not initially recognized as having ACS. The most common symptoms in those patients without chest pain were dyspnea, diaphoresis, nausea and vomiting, and syncope (see Table 12.1) [12]. The mortality rate of patients presenting atypically was 13% as opposed to 4.3% in the typical group. Older adults also have a higher incidence of "silent ischemia." This has been shown in autopsy studies where significant coronary artery stenosis is seen in 50% of patients over the age of 80, although only 20% had demonstrated clinical disease while living [13]. In addition, older adults have longer intervals from time of symptom onset to diagnosis and are more likely to have nondiagnostic electrocardiograms [14]. While younger patients have ST-elevation myocardial infarctions (STEMIs) as their presentation of ACS about 80% of the time, geriatric patients more commonly have non-STEMIs. Only 35% of those older than 84 will have a STEMI as their presentation of ACS (Figure 12.1) [15].

Table 12.1 Most common symptoms that manifest as atypical presentations of acute coronary syndrome (ACS).

Symptom	Percentage of time symptom is seen in cases of "atypical" ACS
Dyspnea	50%
Diaphoresis	26%
Nausea/vomiting	24%
Syncope	19%

Costochondritis is a condition where the costochondral or chondrosternal junction becomes inflamed, causing anterior chest pain and tenderness on palpation. A benign condition, it is usually treated with analgesics, reassurance, and rest. Practitioners often palpate the chest wall of patients presenting with chest pain as part of the physical exam. Tenderness on palpation can be falsely reassuring, prompting a diagnosis of "musculoskeletal" chest wall pain. However, 6–8% of cases of ACS will have chest wall tenderness on examination [16]. In a study where patients were considered to have "noncardiac chest pain" after history, physical examination, and electrocardiogram, nearly 3% went on to have a definite adverse cardiac event, with another 3.5% having a possible adverse cardiac event [17]. Mislabeled patients were an average of 13 years older, with a mean age of 61.1 years. It is with extreme caution that a geriatric patient should be diagnosed as having costochondritis without further evaluation for more serious pathology.

Pitfall | **Minimizing geriatric abdominal pain**

Geriatric patients with abdominal pain are among the most high-risk patients that an acute care provider will encounter. In some studies the mortality rate of an older adult presenting with abdominal pain is higher than a similar patient presenting with chest pain. Yet too often providers trivialize this complaint or attribute symptoms to a lesser illness such as gastroenteritis. The reasons for this are myriad. The elderly patient often presents very atypically, which leads to frequent misdiagnoses. It may be more difficult to take an adequate history due to communication challenges associated with cognitive impairment such as dementia, age-related hearing loss, or simply stoicism. Medications may also mask pathology. As discussed earlier, vital signs may be less reliable in this age group. So too is the physical examination. The abdominal musculature is typically thinner in older patients. This leads to a decrease in rigidity and guarding, even in cases of frank peritonitis. Laboratory

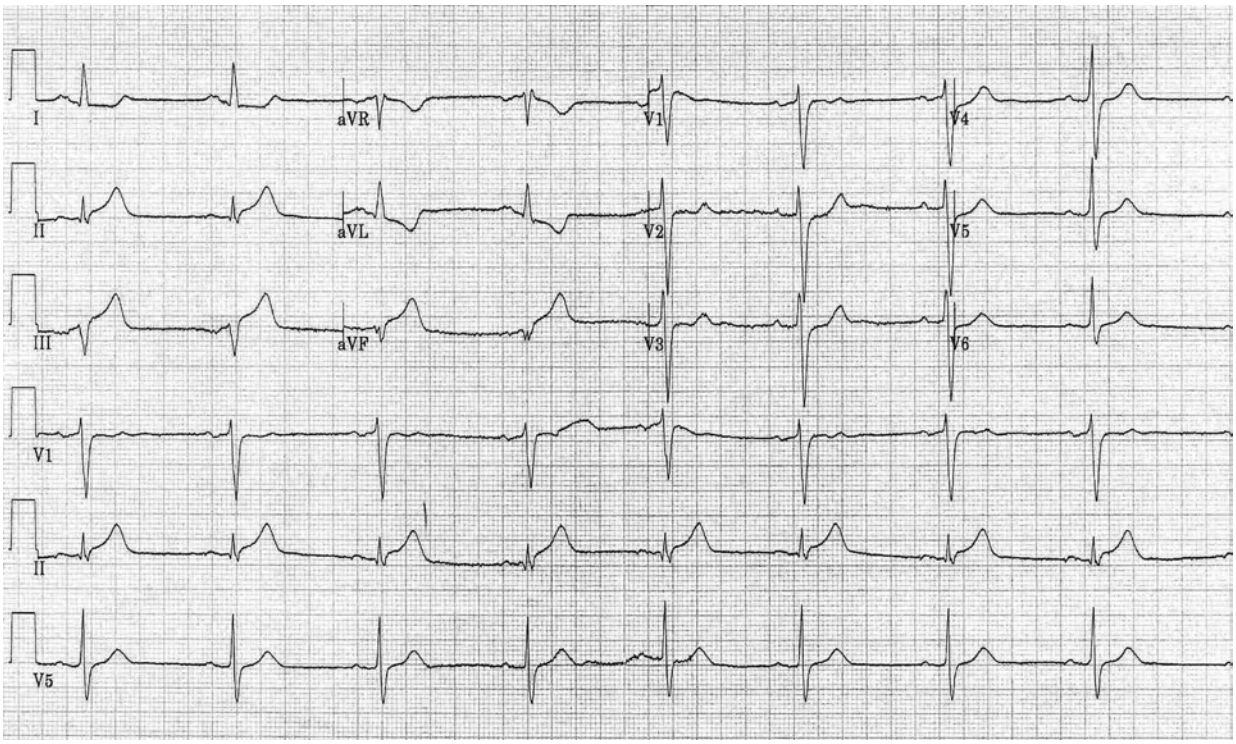

Figure 12.1 Inferior ST-elevation myocardial infarction in an elderly patient who presented with nausea and vomiting but absence of chest pain. Older adults often present without pain during acute coronary syndrome.

values may be falsely reassuring. Nearly one-quarter of cases of appendicitis and 40% of cases of cholecystitis will have normal white blood cell (WBC) counts [18, 19].

Peptic ulcer disease

As previously noted, older adults may have PUD without symptoms. One-half of all ulcers in this age group will have a complication such as a perforation as their presenting symptom [20]. Perforations present very differently in the elderly than in younger patients. Acute onset of pain is found in only 50% of cases. The rigid, board-like abdomen that many expect to find is noted in a minority of cases, roughly 20% [21]. Free intraperitoneal air on plain radiographs is seen in less than one-half of cases (see Figure 12.2) [22]. Elderly patients fare worse than younger patients with conservative treatment [23]. They are more likely to have serious bleeding that requires transfusion or operative management [24]. All of these factors lead to a mortality rate from PUD three times as high in the geriatric population compared to younger patients [25].

KEY FACT | **Less than one-quarter of cases of perforated viscus will present with frank rigidity.**

Biliary tract disease

Biliary tract disease is the most common reason for acute abdominal surgery in older adults, and it can also be challenging to diagnose. While most geriatric patients will present with tenderness in the right upper quadrant or epigastrium, other supporting evidence may be absent. More than half of older adults with acute cholecystitis will not have nausea or vomiting and half will be afebrile [19]. WBC counts and liver function tests are frequently normal. While ultrasound remains a reasonable screening test, older adults are at higher risk for acalculous cholecystitis, which may be better appreciated on a radionuclide scan.

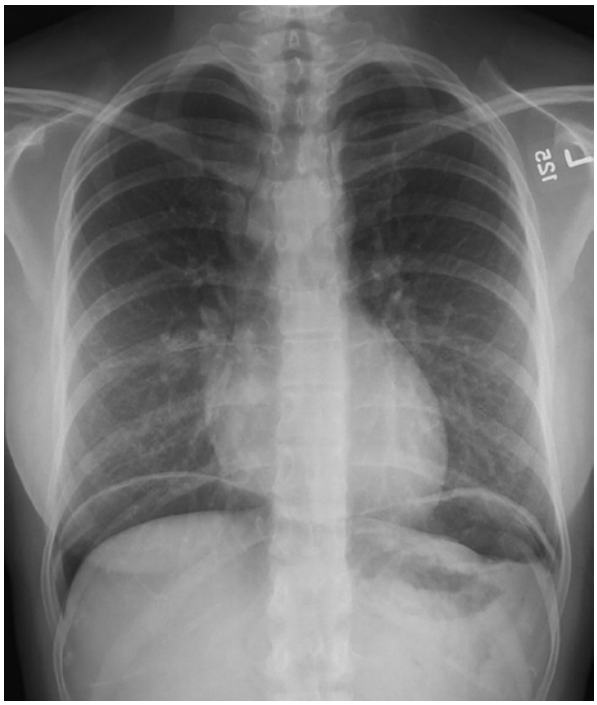

Figure 12.2 Free intraperitoneal air in a patient with a perforated gastric ulcer. Of note, only 40% of the elderly with a perforated viscus will have free air on plain radiographs.

Choledocholithiasis is also more common in older adults and with it ascending cholangitis. Acute suppurative cholangitis is rarely seen before the seventh decade. This diagnosis mandates emergent decompression, either through surgery, endoscopy, or percutaneous drainage.

Appendicitis

Acute appendicitis is the third most common indication for abdominal surgery in older adults [26]. Despite its lower incidence in this population, geriatric patients account for 50% of all deaths from appendicitis, in part due to difficulty in diagnosis [27]. Older patients tend to present later, with one-fifth presenting after three days of symptoms and another 5–10% after one week of symptoms [28]. Only 20% of geriatric patients will present with classic findings of fever, right lower quadrant (RLQ) pain, and leukocytosis. Fever is absent in two-thirds, RLQ tenderness is absent in 30%, and

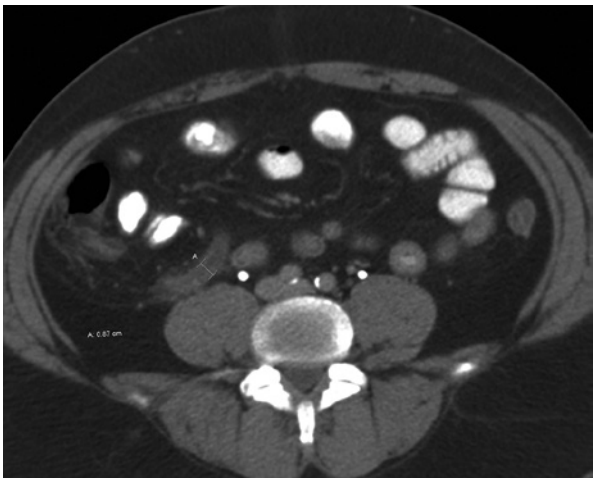

Figure 12.3 A dilated appendix that is not filling with oral contrast, consistent with acute appendicitis.

leukocytosis is absent in one-quarter. One-quarter of all cases of appendicitis in older adults are initially misdiagnosed and discharged home [29]. It is not surprising then that mortality rates are 4–8% in this population (compared to 1% in younger patients) and perforation rates are a staggering 70% [30]. Liberal use of computed tomography (CT) scanning is encouraged in this age group if the patient still possesses an appendix (see Figure 12.3).

KEY FACT | **One-quarter of all cases of acute appendicitis in the elderly are missed at first presentation to an acute care provider.**

Mesenteric ischemia

Acute mesenteric ischemia (AMI) is a life-threatening disorder resulting in hypoperfusion of the gut. Causes include embolism to the superior mesenteric artery (SMA), thrombosis of the SMA, mesenteric venous thrombosis, or a low-flow state (nonocclusive AMI). The majority of cases are due to an SMA embolus. Presentation of AMI includes acute abdominal pain, often out of proportion to the physical examination. "Gut emptying" is frequently seen in cases of mesenteric ischemia. This refers to vomiting, diarrhea, or both and may explain why the most common misdiagnosis of AMI is gastroenteritis. Although atrial fibrillation is the classic source of emboli, in reality it is seen in less than

50% of cases [31]. An important diagnostic clue on history is that one-third of patients with an SMA embolus will have had a prior embolic event, such as an ischemic stroke. Thrombosis of the SMA typically occurs in patients with atherosclerosis risk factors. Patients may relate a history of "intestinal angina" or postprandial abdominal pain. This may cause "food fear" where patients avoid eating and lose significant amounts of weight. Similar to ACS, plaque rupture leads to the acute onset of abdominal pain akin to an SMA embolus. Mesenteric venous thrombosis will often present less acutely, with symptoms lasting days to weeks before diagnosis. These patients may have an underlying hypercoagulable state and half will have a personal or a family history of venous thromboembolic disease. Nonocclusive AMI is seen in low-flow states such as sepsis, dehydration, or severe cardiomyopathy. It may also be seen in hemodialysis patients or patients using digitalis [32].

KEY FACT | **One-half of patients with mesenteric venous thrombosis will have a personal or family history of venous thromboembolic disease.**

The physical examination is often nonspecific in early AMI. Clinicians should not be falsely reassured by a soft abdomen in patients with a concerning history or risk factors for this disease (see Box 12.1). Patients suspected of having AMI should be sent for emergent CT angiography and surgical consultation. Similar to ACS, time is of the essence in these cases. Older adults can have good outcomes and return to their previous quality of life if diagnosed and treated promptly [33].

Box 12.1 Types of mesenteric ischemia and historical clues

SMA embolus
- One-third of cases have had a previous embolic event

SMA thrombosis
- Typical atherosclerosis risk factors
- History of intestinal angina or "food fear"

Mesenteric venous thrombosis
- Presents less acutely
- One in two has a personal or family history of venous thromboembolism

Nonocclusive mesenteric ischemia
- Low-flow states
- Often ICU patients
- Association with digitalis use and hemodialysis

ICU, intensive care unit; SMA, superior mesenteric artery.

Ruptured abdominal aortic aneurysm

Ruptured abdominal aortic aneurysm (AAA) is another imitator in the geriatric population. While the diagnosis may be evident in the patient with hypotension, abdominal pain, and pulsatile abdominal mass, this presentation occurs in a minority of cases. Hypotension is absent in 65% of cases [34]. Pain may be confined to the back or flank instead of the abdomen, and palpable pulsatile masses are often absent or missed on physical examination. As these patients may present with flank pain radiating toward the groin and microscopic hematuria (caused by irritation of the ureter by the enlarging AAA), they may be misdiagnosed as having renal colic. Other frequent misdiagnoses include musculoskeletal back pain and diverticulitis. The misdiagnosis rate is as high as 50% [35, 36]. Geriatric patients presenting with new-onset renal colic symptoms should undergo imaging of their aorta with either ultrasound or CT.

KEY FACT | **Hypotension is absent in 65% of cases of ruptured abdominal aortic aneurysm.**

Urinary tract infections

Pitfall | Overdiagnosis of urinary tract infections is common

Asymptomatic bacteriuria is seen in up to 20% of older female adults living in the community and 50% of those in assisted living or nursing homes [37]. This condition is often identified as acute infection in many older adults with nonspecific symptoms such as general malaise, changes in behavior, and falls and is treated with antibiotics. When typical signs and symptoms of a urinary tract infection (UTI) such as fever, leukocytosis, suprapubic pain, dysuria, and urinary frequency accompany nonspecific symptoms and a positive urinalysis, a diagnosis of UTI may be appropriate. However, in the absence of typical signs and symptoms of infection or UTI, the presence of nonspecific symptoms does not correlate well with a

true UTI [38–40]. Clinicians who prematurely diagnose UTIs in older adults may be experiencing the cognitive error of search-satisfying bias and failing to identify the true underlying cause of a patient's presentation.

Pitfall | **Underappreciating the morbidity and mortality associated with "simple" falls**

The most common etiology of trauma in the elderly is falls [41]. The leading cause of death from injury in patients over the age of 65 is complications from falls with an 11% overall mortality [42]. Most falls, including injurious falls, occur during routine, nonhazardous activities and often while walking on level ground [43].

Mechanisms of injury thought to be "low impact," such as falls from a standing position, can result in significant injuries including fractures due to coexisting osteoporosis, osteopenia, and sarcopenia (loss of skeletal muscle mass).

The aging brain decreases in both size and weight. This leads to stretching of bridging vessels on the surface of the brain, which can then tear from relatively minor shearing forces. Patients may be taking anticoagulation that increases their risk of bleeding. All of these mandate that the acute care provider take falls seriously and perform a thorough evaluation for serious injury.

The cause of the fall must also be investigated. The term "mechanical fall" is often used to describe falls that result from environmental factors such as missed steps, area rugs, and thresholds. However, classifying a fall as mechanical does not signal the absence of an organic process or preventable cause. Visual impairment, loss of peripheral sensation, alcohol use disorders, and polypharmacy should be considered in so-called mechanical falls [44, 45]. Syncope should be considered in unexplained falls, as 20% of patients with a cardiovascular cause of syncope will present merely with unexplained falls [46]. Falls from underlying illnesses including serious entities such as ACS or vascular catastrophes should also be considered. Acute care providers must always be vigilant for the possibility of elder abuse as a cause of "falls."

Central nervous system injuries

As noted already, older adults are at much greater risk for intracranial pathology due to the physiologic changes in brain and bone that accompany aging. The presentation of such pathology in geriatric patients may be occult. Of 1934 patients above the age of 65 in the National Emergency X-Radiography Utilization Study (NEXUS) II trial, 178 (9.2%) had significant intracranial injury and 44% of these patients had no focal neurologic deficits. A small (2.2%) but significant proportion of patients had occult injury, defined as a normal level of consciousness, no neurologic deficit, and no evidence of skull fracture. This presentation was seen in only 0.8% of younger patients [47]. The investigators were "unable to identify criteria that could reliably be used to detect or exclude injury in elders." As such, liberal use of CT imaging is advised in this patient population (see Figure 12.4).

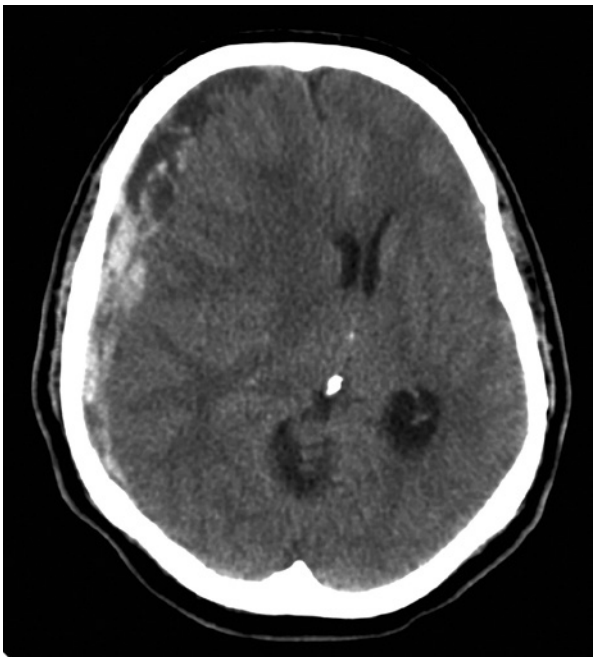

Figure 12.4 Right subdural hematoma with midline shift in an elderly patient on warfarin who presented after a fall from standing.

Intracranial hemorrhage (ICH) is a serious complication of systemic anticoagulant use and is more commonly encountered in older adults. However, evidence-based use of anticoagulants is appropriate for many conditions, also more commonly seen in older adults, including venous thromboembolism, atrial fibrillation, and prostatic heart valves. Even in patients with a history of ICH or at risk for complications from anticoagulant use (such as falls), the benefit of anticoagulants often outweighs the risks [48, 49]. Detailed shared decision-making conversations about the risks and benefits of long-term

anticoagulants are best conducted by a patient's primary care provider. As such, unless there is evidence of active, acute bleeding, the urgent care provider should generally not stop anticoagulants.

Fractures

Fractures complicate 5–10% of all falls in older adults [50]. Forearm fractures are the most common fractures in females, while hip fractures are the most common in males and overall [51]. The presence of hip pain or the inability to ambulate despite negative plain radiographs should prompt consideration of occult hip fracture. Nearly 5% of patients with these complaints and negative plain radiographs will have an occult fracture found on advanced imaging [52]. Patients unable to ambulate after a fall should not be discharged without further evaluation or observation.

Low-level falls lead to increased risk of injury to the spine in the levels between occiput and C2 [53]. One study found that clinical predictors were inadequate for evaluating geriatric patients with low-level injuries [54]. The Canadian C-Spine Rule considers all patients above the age of 65 to be high risk and recommends routine radiographic evaluation in this group [55]. The presence of a cervical spine fracture is associated with increased morbidity and mortality (up to 25%), regardless of whether there is associated spinal cord injury or other traumatic injuries [56].

Nondisplaced rib fractures, better tolerated in younger patients, are a source of significant morbidity and mortality in older adults. The mortality seen in older adults with rib fractures is twice that of younger patients. Even a single rib fracture can lead to significant morbidity. With each successive rib fractured, mortality and the risk of pneumonia increase. Three or more rib fractures in older adults is associated with a 1 in 5 risk of death [57]. Up to 90% of older adults with an acute rib fracture have other traumatic injuries. Given this, geriatric patients found to have acute rib fractures after trauma warrant very thorough evaluation for other injuries and often referral to an ED or trauma center [58, 59].

KEY FACT | **Rib fractures are associated with significant morbidity and mortality in the elderly, and these rates increase with each successive rib fractured.**

Pitfall | **Failing to consider elder abuse and neglect**

While elder abuse and neglect (EAN) may be obvious in some cases, most incidents are more subtle and go unrecognized. First described in the medical community in 1975, EAN has finally become recognized as a distinct entity and is now the subject of much research [60, 61]. Abuse may take many forms: physical, sexual, psychological, or financial as well as neglect, abandonment, exploitation, and violation of personal rights. Abuse can occur in the older adult's domicile or in an institutional setting. Actual numbers are hard to quantify, but it is estimated that at least 3–5% of all elders have been victimized at some point, with the most common type of abuse being neglect [62–64]. By far the most likely perpetrator is a close relative, with adult children committing abuse in nearly half of all cases and a spouse being the abuser in one-fifth [65]. Alcoholism or other substance abuse by the perpetrator is the most predictive characteristic [66, 67].

Most acute care practitioners do not routinely ask older adults about EAN, though it has been shown that this simple act can increase detection. Careful physical examination may uncover clues such as bruises in various stages of healing (especially if not over bony prominences), burns, patterned injuries such as hand slaps or ligature marks, or signs of neglect including infestation or untreated injuries. Radiographs may be warranted and may show signs of acute or remote fractures. A low threshold for neuroimaging should be maintained in cases of suspected abuse, as significant injuries can occur from shaking or low-level falls.

Reporting requirements may vary, and it is crucial to be familiar with local regulations. Regardless of reporting requirements, the safety of an older adult patient should be confirmed prior to discharge.

Conclusion

Older adults often present with atypical signs and symptoms. Vital signs may appear normal and physical examination may be unremarkable despite serious illness and pathology. There are certain diagnoses that the astute practitioner should be particularly cautious with in the elderly population. Costochondritis and gastroenteritis are diagnoses that should give the acute care clinician particular pause when used in older adults. Falls are

very common in this population, as are serious injuries from falls. Every provider who cares for geriatric patients should be vigilant for possible elder abuse.

Pearls for improving patient outcomes

- An electrocardiogram should be obtained early in the care of any elderly patient presenting with vague complaints including unexplained dyspnea, nausea/vomiting, or diaphoresis.
- Asymptomatic hypertension does not require urgent treatment.
- Be aggressive when confronted with abnormal vital signs and do not attribute them simply to pain.
- Before making a urinary tract infection diagnosis, make sure there are both signs and symptoms present of such a condition.
- Consider mesenteric ischemia any time an elderly patient is diagnosed with "gastroenteritis."
- Any elderly patient being diagnosed with a first episode of nephrolithiasis should have their aortic size assessed, with either computed tomography or ultrasound.
- Every geriatric patient who presents with an injury should be screened for elder abuse.

References

1 United States Census Bureau. National population projections tables: main series. 2023. https://www.census.gov/data/tables/2023/demo/popproj/2023-summary-tables.html. Accessed December 24, 2023.

2 Our World in Data. Population by age group, world. 2022. https://ourworldindata.org/grapher/population-by-age-group-with-projections. Accessed December 24, 2023.

3 Fenyo G. Diagnostic problems of acute abdominal diseases in the aged. *Acta Chir Scand* 1974; **140**: 396–405.

4 Heffernan DS, Thakkar RK, Monaghan SF, *et al*. Normal presenting vital signs are unreliable in geriatric blunt trauma victims. *J Trauma* 2010; **69**: 813–820.

5 Chobanian AV, Bakris GL, Black HR, *et al*. Seventh report of the joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. *Hypertension* 2003; **42**: 1206–1252.

6 Scalea T, Simon H, Duncan A, *et al*. Geriatric blunt multiple trauma: improved survival with early invasive monitoring. *J Trauma* 1990; **30**: 129–134.

7 Edwards M, Let E, Mirocha J, *et al*. Defining hypotension in moderate to severely injured trauma patients: raising the bar for the elderly. *Am Surg* 2010; **76**: 1035–1038.

8 Sherman ED, Robillard E. Sensitivity to pain in the aged. *Can Med Assoc J* 1960; **83**: 944–947.

9 Leverat M. Peptic ulcer disease in patients over 60: experience in 287 cases. *Am J Dig Dis* 1966; **11**: 279–285.

10 Patel KK, Young L, Howell EH, *et al*. Characteristics and outcomes of patients presenting with hypertensive urgency in the office setting. *JAMA Intern Med* 2016; **176**: 981–988.

11 Bhatia LC, Naik RH. Clinical profile of acute myocardial infarction in elderly patients. *J Cardiovasc Dis Res* 2013; **4**(2): 107–111.

12 Brieger D, Eagle K, Goodman S, *et al*. Acute coronary syndromes without chest pain, an underdiagnosed and undertreated high-risk group. *Chest* 2004; **126**: 461–469.

13 Elveback L, Lie JT. Continued high incidence of coronary artery disease at autopsy in Olmsted County, Minnesota, 1950 to 1979. *Circulation* 1984; **70**: 345–349.

14 Kyriades ZS, Kourouklis S, Kontaras K. Acute coronary syndromes in the elderly. *Drugs Aging* 2007; **24**(11): 901–912.

15 Cheitlin MD, Zipes DP. Cardiovascular disease in the elderly. In: Braunwald E, Zipes DP, Libby P (eds.), *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine* (6th edn.). Philadelphia (PA): Saunders; 2001: pp. 2019–2037.

16 Disla E, Rhim HR, Reddy A, *et al*. Costochondritis. A prospective analysis in an emergency department setting. *Arch Intern Med* 1994; **154**(21): 2466–2469.

17 Miller CD, Lindsell CJ, Khandelwal S, *et al*. Is the initial diagnostic impression of "noncardiac chest pain" adequate to exclude cardiac disease? *Ann Emerg Med* 2004; **44**(6): 565–574. Correction *Ann Emerg Med* 2005; **45**(1):87.

18 Horattas MC, Guyton DP, Wu D. A reappraisal of appendicitis in the elderly. *Am J Surg* 1990; **160**: 291–293.

19 Morrow DJ, Thompson J, Wilson SE. Acute cholecystitis in the elderly. *Arch Surg* 1978; **113**: 1149–1152.

20 Caesar R. Dangerous complaints: the acute geriatric abdomen. *Emerg Med Rep* 1994; **15**: 191–202.

21 Fenyo G. Acute abdominal disease in the elderly: experience from two series in Stockholm. *Am J Surg* 1982; **143**: 751–754.

22 McNamara RM. Acute abdominal pain. In: Sanders AB (ed.), *Emergency Care of the Elder Person*. St. Louis (MO): Beverly Cracom Publications; 1996: pp. 219–243.

23 Crofts TJ, Park KG, Steele RJ. A randomized trial of nonoperative treatment for perforated peptic ulcer. *N Engl J Med* 1989; **320**(15): 970–973.

24 Borum ML. Peptic-ulcer disease in the elderly. *Clin Geriatr Med* 1999; **15**: 457–471.

25 Wakayama T. Risk factors influencing the short-term results of gastroduodenal perforation. *Surg Today* 1994; **24**(8): 681–687.

26 Kauvar DR. The geriatric acute abdomen. *Clin Geriatr Med* 1993; **9**: 547–545.

27 Shoji BT, Becker JM. Colorectal disease in the elderly patient. *Surg Clin N Am* 1994; **74**: 293–316.

28 Freund HR, Rubinstein E. Appendicitis in the aged: is it really different? *Am Surg* 1984; **50**: 573–576.

29 Storm-Dickerson TL, Horratas MC. What have we learned over the past 20 years about appendicitis in the elderly? *Am J Surg* 2003; **185**: 198–201.

30 Yamini D, Vargas H, Bongard F, *et al*. Perforated appendicitis: is it truly a surgical urgency? *Am Surg* 1998; **64**: 970–975.

31 Park WM, Gloviczki P, Cherry KJ Jr, *et al*. Contemporary management of acute mesenteric ischemia: factors associated with survival. *J Vasc Surg* 2002; **35**(3): 445–452.

32 Diamond S, Emmett M, Henrich WL. Bowel infarction as a cause of death in dialysis patients. *JAMA* 1986; **256**: 2545–2547.

33 Pengermä P, Venesmaa S, Karjalainen J, *et al*. Long-term outcome after implementation of endovascular-first strategy to treat acute mesenteric ischemia. *J Vasc Surg* 2023; **78**(6): 1524–1530. https://doi.org/10.1016/j.jvs.2023.08.100.

34 Rutherford RB, McCroskey BL. Ruptured abdominal aneurysm: special considerations. *Surg Clin North Am* 1989; **69**: 859–868.

35 Marston WA, Ahlquist R, Johnson G Jr, *et al*. Misdiagnosis of ruptured abdominal aortic aneurysms. *J Vasc Surg* 1992; **16**: 17–22.

36 Salkin MS. Abdominal aortic aneurysm: avoiding failure to diagnose. *ED Leg Lett* 1997; **8**: 67–78.

37 Nicolle LE. Asymptomatic bacteriuria. *Curr Opin Infect Dis* 2014; **27**(1): 90–96.

38 Caterino JM, Kline DM, Leininger R, *et al*. Nonspecific symptoms lack diagnostic accuracy for infection in older patients in the emergency department. *J Am Geriatr Soc* 2019; **67**(3): 484–492. https://doi.org/10.1111/jgs.15679.

39 Frazee BW, Enriquez K, Ng V, *et al*. Abnormal urinalysis results are common, regardless of specimen collection technique, in women without urinary tract infections. *J Emerg Med* 2015; **48**(6): 706–711.

40 Goebel MC, Trautner BW, Grigoryan L. The five Ds of outpatient antibiotic stewardship for urinary tract infections. *Clin Microbiol Rev* 2021; **34**(4): e0000320.

41 Aschkenasy MT, Rothenhaus TC. Trauma and falls in the elderly. *Emerg Med Clin North Am* 2006; **24**: 413–432.

42 Mosenthal AC, Livingston DH, Elcavage J, *et al*. Falls: epidemiology and strategies for prevention. *J Trauma* 1995; **38**: 752–756.

43 Talbot LA, Musiol RJ, Witham EK, *et al*. Falls in young, middle-aged and older community dwelling adults: perceived cause, environmental factors and injury. *BMC Public Health* 2005; **5**: 86–86.

44 Sri-on J, Tirrell GP, Lipsitz LA, *et al*. Is there such a thing as a mechanical fall? *Am J Emerg Med* 2016; **34**(3): 582–585. https://doi.org/10.1016/j.ajem.2015.12.009.

45 O'Connell H, Chin AV, Cunningham C, *et al*. Alcohol use disorders in elderly people – redefining an age old problem in old age. *BMJ* 2003; **327**: 664–667.

46 Mitchell LE, Richardson DA, Davies AJ, *et al*. Prevalence of hypotensive disorders in older patients with a pacemaker in situ who attend the accident and emergency department because of falls or syncope. *Europace* 2002; **4**: 143–147.

47 Rathlev NK, Medzon R, Lowery D, *et al*. Intracranial pathology in elders with blunt head trauma. *Acad Emerg Med* 2006; **13**: 302–307.

48 Kuramatsu JB, Huttner HB. Management of oral anticoagulation after intracerebral hemorrhage. *Int J Stroke* 2019; **14**(3): 238–246. https://doi.org/10.1177/1747493019828555.

49 Li YG, Lip GYH. Anticoagulation resumption after intracerebral hemorrhage. *Curr Atheroscler Rep* 2018; **20**(7): 32. https://doi.org/10.1007/s11883-018-0733-y.

50 van Weel C, Vermeulen H, van den Bosch W. Falls, a community care perspective. *Lancet* 1995; **345**: 1549–1551.

51 Nordell E, Jarnlo G, Jetsen C, *et al*. Accidental falls and related fractures; a retrospective study of 332 patients. *Acta Orthop Scand* 2000; **71**(2): 175–179.

52 Dominguez S, Liu P, Roberts C, *et al*. Prevalence of traumatic hip and pelvic fractures in patients with suspected hip fracture and negative initial standard radiographs a study of emergency department patients. *Acad Emerg Med* 2005; **12**(4): 366–369.

53 Sharma OP, Oswanski MF, Yazdi JS, *et al*. Assessment for additional spinal trauma in patients with cervical spine injury. *Am Surg* 2007; **73**(1): 70–74.

54 Schrag SP, Toedter LJ, McQuay N. Cervical spine fractures in geriatric blunt trauma patients with low energy mechanism: are clinical predictors adequate? *Am J Surg* 2008; **195**: 170–173.

55 Stiell IG, Wells GA, Vandemheen KL, *et al*. The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 2001; **286**: 1841–1848.

56 Golob JF, Claridge JA, Yowler CJ, *et al*. Isolated cervical spine fractures in the elderly: a deadly injury. *J Trauma* 2008; **64**: 311–315.

57 Bulger EM, Arneson MA, Mock CN, *et al*. Rib fractures in the elderly. *J Trauma* 2000; **48**(6): 1040–1047.

58 Coary R, Skerritt C, Carey A, *et al*. New horizons in rib fracture management in the older adult. *Age Ageing* 2020; **49**(2): 161–167.

59 Ziegler DW, Agarwal NN. The morbidity and mortality of rib fractures. *J Trauma* 1994; **37**(6): 975–979.

60 Burston GR. Granny-battering. *BMJ* 1975; **3**: 592.

61 Baker AA. Granny battering. *Mod Geriatr* 1975; **5**: 20–24.

62 Pillemer K, Finkelhor D. The prevalence of elder abuse: a random sample survey. *Gerontologist* 1988; **29**: 51–57.

63 Davidson JL. Elder abuse. In: Block MR, Sinnott JD (eds.), *The Battered Elder Syndrome: An Exploratory Study*. College Park (MD): University of Maryland; 1979: pp. 49–66.
64 Lachs MS, Williams C, O'Brien S, *et al*. Risk factors for reported elder abuse and neglect: a nine-year observational cohort study. *Gerontologist* 1997; **37**: 469–474.
65 United States Department of Health and Human Services Administration on Aging and the Administration for Children and Families. *The National Elder Abuse Incidence Study*. Washington (DC): NCEA; 1998.
66 Kosberg JI, Nahmiash D. Characteristics of victims and perpetrators and milieus of abuse and neglect. In: Baumhover LA, Beall SC (eds.), *Abuse, Neglect, and Exploitation of Older Persons: Strategies for Assessment and Intervention*. Baltimore (MD): Health Professions Press; 1996: pp. 31–49.
67 Lachs MS, Pillemer K. Abuse and neglect of elderly persons. *N Engl J Med* 1995; **332**: 437–443.

CHAPTER 13
Pharmacology Pitfalls and Pearls in Urgent Care Medicine

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Introduction

The US Food and Drug Administration Adverse Events Reporting System reported over 190,000 deaths in 2020 due to medication adverse events or errors, making medication-related problems the fifth leading cause of death in the United States [1, 2]. Adverse drug events account for 17 million emergency department visits in the United States annually. Costs associated with adverse drug events totaled $177 billion in 2000. A recent study revealed that 12% of visits to a tertiary emergency department were drug related and 68% of these events were preventable. Preventable adverse drug events involve incorrect dosing; a known allergy; a known interaction; nonadherence; lack of laboratory monitoring; and prescribing, dispensing, or administrative errors [3]. Furthermore, several medications can have side effects that cause unneeded anxiety among patients and providers. Knowledge of these common adverse effects can prevent anxiety by providing patients with proper anticipatory guidance. Finally, certain populations need special consideration to mitigate medication pitfalls.

Pitfalls and pearls in pharmacology of commonly used medications

Analgesics

Nonsteroidal anti-inflammatory drugs

Pitfall | **Failing to recognize patients at risk for gastrointestinal and renal complications from nonsteroidal anti-inflammatory drugs**

Nonsteroidal anti-inflammatory drugs (NSAIDs) work by inhibiting one or both cyclooxygenase (COX) enzymes. COX enzyme inhibition results in both untoward and therapeutic NSAID effects. In the kidney, COX-1 produces prostacyclins that maintain renal blood flow. In the gastrointestinal tract, COX-1 produces a prostacyclin that enhances mucosal perfusion, bicarbonate production, and mucous production, which contribute to mucosal integrity. Depletion of prostaglandins impairs the structural integrity of the mucosal cells resulting in ulcer formation. NSAIDs are the second most common cause of peptic ulcer formation behind *Helicobacter pylori* infection [4].

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There are variable safety profiles among NSAIDs. Ibuprofen is recognized as one of the safest, with no gastrointestinal effect with up to seven days of use. Ketorolac, however, should not be used for more than five days in order to reduce the risk of acute renal failure. Care should still be taken when recommending or prescribing NSAIDs to individuals with chronic renal impairment to avoid additional nephrotoxicity [5].

Acetaminophen

Pitfall | **Failing to recognize products that contain acetaminophen**

More than 200 medication combinations contain acetaminophen as a component. Typical recommendations for maximal daily dosage are 3 g/day in adults and 75 mg/kg/day in children. A small amount of acetaminophen is normally metabolized by the liver into a highly hepatotoxic metabolite. Normally, protective mechanisms rapidly metabolize these toxic products. With excessive acetaminophen ingestion these can be overwhelmed, resulting in hepatotoxicity. Usually, a 7.5 g or 150 mg/kg or greater dose is required to cause hepatotoxicity. Patients with liver dysfunction or those with chronic supratherapeutic ingestions are at greater risk for toxicity. Chronic overdose or usual doses in patients with liver dysfunction can cause toxicity. Many chronic ingestions can be inadvertent, as many combination preparations contain acetaminophen. Opioid–acetaminophen combinations exist as well as over-the-counter combinations with NSAIDs, sedatives, cough and cold medications, and other agents (e.g. Midrin®, Excedrin®, etc.). If acute acetaminophen toxicity is suspected, a serum level should be obtained four hours after the time of ingestion (in cases of acute ingestion) and plotted on the Rumack–Matthew nomogram to determine whether n-acetylcysteine antidote therapy is required [6]. The Rumack–Matthew nomogram does not apply to chronic overdose. A patient should be treated for acetaminophen toxicity if chronic overdose is suspected and liver function tests are abnormal [7]. Consultation with a toxicologist or poison control center is strongly recommended if patients with suspected toxicity are not transferred to a higher level of care.

KEY FACT | **Over-the-counter analgesics can cause significant toxicity, some of which may have subtle presentations.**

Multimodal analgesia

Pitfall | **Failing to utilize multimodal analgesia**

The treatment of pain in acute care settings is subject to fluctuations in prioritization and balancing the risks and benefits of different treatment modalities. Treatment should be tailored to the underlying cause of pain and consideration of different approaches to analgesia should take place. Potential interventions include oral or parenteral acetaminophen and/or NSAIDs, local infiltration of anesthetic, topical administration of various medications, nerve block, hematoma block, and opioids.

For most minor injuries, acetaminophen and NSAIDs are the first-line treatment for pain. Additionally, there is a synergistic effect with use of acetaminophen in combination with other analgesics (opioid or NSAID) [8].

NSAIDs have the added benefit of providing reduction of inflammation at higher doses (2400 mg/day divided to 600–800 mg doses) [9, 10], which can address some of the underlying causes of pain. When used in combination, acetaminophen plus NSAIDs can provide pain relief via different receptor pathways, resulting in improved effect.

Topical preparations of NSAIDs are available and can provide benefit to patients who are unable to tolerate systemic treatment due to underlying medical conditions.

Lidocaine patches are available over the counter at reasonable cost (4%) or with a prescription (5%) and can be directly applied to closed skin in the area of pain.

If a compounding pharmacy is available in the area, a topical cream containing amytriptyline and ketamine may be used, particularly in cases of neuropathic pain [11].

Dental pain is a common presenting complaint and in addition to treating the cause, an approach that utilizes both anti-inflammatory and anesthetic can be beneficial. Use of a bupivacaine dental block can provide several hours of relief, potentially bridging to definitive care by a dentist.

Opioids

When opioids are indicated, they should be used in combination with nonopioid analgesics as part of a multimodal strategy and should be prescribed for the shortest reasonable duration (Table 13.1).

Disparities in opioid prescribing practices are persistent despite documentation of race- and gender-based disparities in analgesia administration for decades [13].

Table 13.1 Opioid analgesic dosing.

	Dose equivalent to morphine 10 mg IM		
Agent	Parenteral	Oral	Recommended initial dose[a]
Morphine sulfate	10 mg	30 mg	0.1 mg/kg IV q 1–2 h
Hydrocodone	NA	30–45 mg	5–10 mg PO q 4–6 h
Hydromorphone	2 mg	8 mg	0.5 mg IV q 2–3 h
Oxycodone	NA	20–30 mg	5–10 mg PO q 4–6 h
Fentanyl	0.1 mg	NA	1 μg/kg IV q 30–60 min

IM, intramuscularly; IV, intravenously; NA, not applicable; PO, orally; q, every.
[a] Appropriate opioid dosing can vary widely. The lowest adequate dose should be used. Patient factors such as tolerance, comorbidities (e.g. renal function, hepatic function), and concurrent adjunctive therapies should be taken into account when dosing opioids.
Source: Data from Micromedex® Healthcare Series [6] and PDQ Supportive and Palliative Care Editorial Board [12].

Providers should strive to appropriately and adequately manage pain. Patients should be asked to rate their level of pain, and treatment should be tailored to meet their needs using a multimodal approach.

Particular care should be taken when caring for patients with chronic or acute-on-chronic back pain. There is little evidence of functional improvement for these patients [14] and the risks of opioid side effects and misuse likely outweigh the benefit.

Local anesthetics

Pitfall | Failing to recognize the potential catastrophic effects of overdose and intravascular injection of local anesthetics

Overdose or intravascular injection of local anesthetics can cause severe side effects including seizures and arrhythmias. Central nervous system intoxication is characterized by muscle twitching followed by generalized tonic–clonic seizure activity. In massive overdose, apnea and coma are seen. Arrhythmias and contractile dysfunction are manifestations of local anesthetic cardiovascular toxicity. A centrally mediated mechanism may also produce bradycardia and hypotension [15].

Awareness of the maximum safe dose for a local anesthetic will prevent toxicity. Special attention should be paid when using local anesthetic for large injuries, when using topical formulation (e.g. 4% lidocaine) or when repeat dosing is needed. Intravascular injection can quickly lead to toxic levels. Aspiration of the needle prior to injection is 98% effective at identifying intravascular placement [16]. Local anesthetics can be combined with epinephrine to improve hemostasis, increase duration of action, and raise the toxic threshold [17].

Pitfall | Failing to differentiate classes of local anesthetics when an allergy to a local anesthetic exists

There are two chemical classes of local anesthetics: amides and esters (Table 13.2). Patients with allergies to an agent are typically allergic to all agents of the same group but may safely be given an anesthetic of the other group. In the rare case that allergy exists to both amides and esters, diphenhydramine 1% can be used as an alternative local anesthetic for wound repair [21].

Pitfall | Failing to minimize the pain of injection of local anesthetics

Local anesthetics have low pH and cause pain on injection. Numerous studies have evaluated the effects of buffering, warming, and rate of injection on pain created by the injection itself [22–24]. Addition of 8.4% sodium bicarbonate can reduce time to onset and decrease pain with injection by raising pH. The amount of 8.4% sodium bicarbonate should not exceed 1 ml per 10 ml of lidocaine. Use of sodium bicarbonate to buffer bupivacaine should be undertaken with caution due to

Table 13.2 Local anesthetics.

	Maximum dose	Notes
Amides		
Lidocaine	4.5 mg/kg; 7 mg/kg with epinephrine	A 1% solution contains 10 mg/ml
Bupivacaine	2 mg/kg; 3 mg/kg with epinephrine	
Mepivacaine	4 mg/kg	Only supplied with epinephrine
Prilocaine and lidocaine cream (EMLA)	–	For use on intact skin Apply under a semi-occlusive dressing Variable systemic absorption
Lidocaine (4%), epinephrine (0.1%), and tetracaine (0.5%) (LET)	–	For use on open skin Variable systemic absorption
Esters		
Procaine	7 mg/kg; 9 mg/kg with epinephrine	–
Chloroprocaine	8 mg/kg	–
Cocaine	150 mg for a 70 kg adult	Used only in the nose, nasopharynx, mouth, throat, and ear
Benzocaine spray (20% solution)	–	Potential to cause methemoglobinemia
Proparacaine	1 drop at a time until adequate anesthesia is obtained	For ophthalmologic use

Source: Data from Todd *et al*. [17]; Tran and Koo [18]; Oni *et al*. [19]; Mills *et al*. [20].

the risk of precipitation. A volume of 0.05 ml sodium bicarbonate in 10 ml 0.5% bupivacaine has been successfully used without precipitation. Warming and a slower rate of injection also decrease pain on injection [25]. Attempts should be made to use all three strategies when administering local anesthetics.

Topical adjuncts to reduce pain of, or eliminate need for, injections can also be implemented. Use of a topical lidocaine gel (lidocaine 4%–epinephrine 0.1%–tetracaine 0.5%) may provide sufficient analgesia [26], allowing avoidance of infiltration and potential tissue deformation when performing a laceration repair. Forgoing injection can be particularly beneficial in children or individuals with increased anxiety. Benzocaine 20% gel is available for intraoral use prior to dental block administration to improve comfort [27].

KEY FACT | **Attention to pain management is an important aspect of emergency care. Care must be taken to use analgesic medications in the most effective and safest way.**

Antiemetics

Pitfall | Administering antiemetics to patients with QTc prolongation

Prescribers should be familiar with the potential for drug-induced ventricular arrhythmias. Certain medications are known to prolong the electrocardiogram (ECG) QT interval, which is a risk factor for developing tachyarrhythmias. Medications from several classes—including antiemetics, antiarrhythmics, antibacterials, and antipsychotics—carry a risk of drug-induced ventricular arrhythmias [28].

Antiemetics are a particularly problematic group of medications that may provoke QTc prolongation. Electrolyte imbalances that may be related to change in oral intake and increased losses may contribute to increased risk of QT prolongation [29]. Ondansetron, which works centrally and peripherally to decrease nausea, is one of the most well known. It lacks many of the side effects (e.g. akathisia) associated with other antiemetics [30] and is available in intravenous, tablet and liquid, and oral-dissolving preparations. However,

Box 13.1 Medications commonly prescribed in urgent care settings that may cause prolonged QTc

Known risk for torsades de pointes:
- Azithromycin
- Ciprofloxacin
- Levofloxacin
- Ondansetron

Possible risk of torsades de pointes:
- Tramadol

Source: Adapted from https://crediblemeds.org/druglist.

other antiemetics including prochlorperazine, promethazine, droperidol, and meclizine also produce QT prolongation. These medications should be avoided in patients with a prolonged QTc interval or a congenital long QT syndrome regardless of current ECG. Ondansetron has been shown to increase the QTc interval by approximately 17 ms and has been associated with the induction of fatal ventricular tachycardia [31–34]. Commonly prescribed medications that may cause prolonged QTc are included in Box 13.1.

The following are steps to prevent ventricular arrhythmia secondary to QTc prolongation:

- If a patient's baseline ECG demonstrates QTc prolongation (>450 ms in males and >470 ms in females) or there is a known history of congenital long QT syndrome, avoid QTc-prolonging agents.
- If a patient does not have a baseline ECG available, be mindful of items in the patient's history that might suggest QTc prolongation. These include a history of syncope or arrhythmia, ischemic heart disease, cardiomyopathy, or congestive heart failure. If a QTc-prolonging agent is being considered, obtain a screening ECG prior to administration.
- If a patient is taking or is starting a medication that is known to prolong the QT interval, consider using another agent or obtaining an ECG to ensure that the patient does not have baseline QT prolongation. Common medications that prolong the QT interval are listed in Box 13.2. Avoid combinations of drugs that are known to prolong the QT interval. Lists of QTc-prolonging medications can be readily found online.
- Review current medications and if a QTc-prolonging drug is already being used, avoid medications that cause hypokalemia or hypomagnesemia, or agents that inhibit the drug's metabolism [34].

Alternative therapies for treatment of nausea in patients with prolonged QT or congenital long QT syndrome are limited. Lorazepam has had a role in antiemetic regimens without generating QT prolongation, but its role in the urgent care setting is likely limited and use as an outpatient is further complicated by risk of misuse.

Complementary and alternative treatments have been studied for their efficacy in treating nausea, largely in the setting of chemotherapy. There is no good evidence to suggest that most of these therapies (acupressure, aromatherapy, music therapy, massage, reflexology, and hypnosis) are effective in reducing nausea. Acupressure/electropressure, guided imagery, or nutritional consultation with a dietician may demonstrate improvement in symptoms. However, this may not be practical in the urgent care setting and is likely influenced by the underlying cause (chemotherapy versus viral illness, for example) [35].

Isopropyl alcohol inhalation has been used as an alternative therapy for nausea. However, its use is not supported by evidence, though the risk of harm is low [36].

Antibiotics

Pitfall | Failing to consider the risks and benefits of prescribing antibiotics

Of the top 20 medications prescribed in an urgent care setting, 40% are antibiotics [37]. Between 80 and 90% of all antibiotics given to humans are prescribed in the outpatient setting and an estimated 30% are unnecessary, while 50% are inappropriate or unnecessary [38]. The reduction of outpatient antimicrobial prescribing is a priority for national-level stewardship efforts [39].

A focused approach to antimicrobial stewardship involves identifying the source and obtaining source control if possible, then selecting the right drug for the right diagnosis, at the right dose for the right duration, and deescalating as appropriate (also known as the 5 Ds of stewardship) [39]. In practice this can be done by choosing the most appropriate therapy for each suspected infection, for an appropriate duration; specific choice of antibiotics for common infection types should be driven by local susceptibility data. The most common infections seen in an urgent care setting are upper respiratory tract infections (URTIs) and urinary tract infections [40]. Many patients with URTIs expect to be

Box 13.2 Common medications that may prolong QT and/or should be avoided in congenital long QT syndrome

Known risk for torsades de pointes:
- Amiodarone
- Azithromycin
- Ciprofloxacin
- Citalopram
- Clarithromycin
- Erythromycin
- Escitalopram
- Fluconazole
- Levofloxacin
- Methadone
- Moxifloxacin
- Ondansetron

Possible risk of torsades de pointes:
- Buprenorphine
- Levetiracetam
- Lithium
- Promethazine
- Tramadol
- Venlafaxine

Conditional risk of torsades de pointes:
- Cimetidine
- Diltiazem
- Diphenhydramine
- Esomeprazole
- Famotidine
- Fluoxetine
- Furosemide
- Hydrochlorothiazide
- Hydroxyzine
- Ketoconazole
- Lansoprazole
- Loperamide
- Metoclopramide
- Metronidazole
- Olanzapine
- Omeprazole
- Pantoprazole
- Paroxetine
- Quetiapine
- Risperidone
- Sertraline
- Torsemide
- Trazodone

Source: Adapted from https://crediblemeds.org/druglist.

prescribed an antibiotic, and this expectation has been shown to increase antibiotic prescriptions [41]. The use of educational materials for patients and providers, including awareness of viral versus bacterial infections and the potential risks of unnecessary antibiotics, has decreased prescribing in the last 10 years [42]. It has also been shown that withholding antibiotics does not decrease patient satisfaction [43, 44].

URTIs can be broken into three tiers. Tier I URTI includes pneumonia and should always have antibiotics prescribed. Tier II URTI includes sinusitis, pharyngitis, suppurative otitis media, and chronic obstructive pulmonary disease (COPD) exacerbation and may or may not have antibiotics prescribed. Infections considered tier III URTI are bronchitis, serous otitis media, viral URTI, influenza, allergy, and asthma and should never have antibiotics prescribed [45]. Tiers II and III can have viral, bacterial, or mixed etiology and are generally self-limited diseases.

In group A streptococcal pharyngitis, antibiotics reduce the incidence of acute rheumatic fever by two-thirds; however, total symptom duration is only shortened by 16 hours. Treatment of only proven (or highly suspected based on diagnostic criteria) group A streptococcal pharyngitis is important to avoid the development of antibiotic resistance. Evidence of the efficacy of antibiotics for acute sinusitis is mixed. Guidelines recommend antibiotics only for patients with moderate to severe symptoms and symptomatic treatment and reassurance for those (most patients) with mild disease [46]. In acute otitis media, antibiotics reduce pain but have no impact on hearing and rarely shorten the course of disease. As these conditions are typically self-limited, a "watch-and-wait" strategy for antibiotics may be appropriate for populations with poor access to healthcare or who have not improved with conservative therapy. The use of watch-and-wait prescriptions through a shared decision model, particularly in the parents of pediatric patients with acute otitis media, results in a significant decrease in unnecessary prescriptions [42, 44, 47]. Benefits of withholding antibiotics include decreased diarrhea, vomiting, rash, and antibiotic resistance [44].

The widespread use of antibiotics has led to unpleasant and sometimes lethal side effects. For instance, the antibiotic-associated intestinal infections caused by *Clostridioides difficile* range from diarrhea to life-threatening colitis. Infection with this toxin-producing organism is becoming progressively more frequent, more severe, more refractory to standard therapy, and more likely to relapse. *C. difficile* is now the most common bacterial cause of diarrhea and approximately 35% of all adult *C. difficile* infections in the United States are community associated [48]. Risk factors include antibiotic exposure, advanced age, and hospitalization. Clindamycin, ampicillin, cephalosporins, and fluoroquinolones are most frequently implicated, but almost any antibiotic can induce disease [49]. Common presenting complaints include diarrhea, abdominal cramps, and fever. Fecal leukocytes are seen and leukocytosis is usually present. Disease severity can range from diarrhea to fulminant

pseudomembranous colitis, which can lead to toxic megacolon, shock, and potentially death. Antibiotic restraint is essential to minimize the risk of this potentially deadly disease [50, 51].

Another undesirable consequence of antibiotic overuse is the accelerated emergence of bacterial resistance. New strains of bacteria resistant to multiple classes of antibiotics (i.e. "superbugs") are steadily increasing. In the past, the problem of resistance was primarily confined to hospitals and nursing homes. However, more recently resistant bacteria have been recognized as a significant cause of community-acquired infections [51]. Methicillin-resistant *Staphylococcus aureus* (MRSA) is an increasingly important cause of skin and soft tissue infections and pneumonia. Fortunately, most strains of community-acquired MRSA are susceptible to trimethoprim-sulfamethoxazole, doxycycline, or clindamycin, depending on local susceptibility patterns [51]. The prevalence of other antibiotic-resistant community-acquired infections is also increasing. In 2019, the US Centers for Disease Control updated its list of Antimicrobial Resistance Threats. Bacteria of concern in a community setting include *C. difficile,* drug-resistant *Neisseria gonorrhoeae* and *Mycoplasma genitalium*, extended-spectrum beta-lactamase–producing *Enterobacterales*, MRSA, and drug-resistant *Streptococcus pneumoniae* [51].

Cephalosporins

Pitfall | **Failing to categorize the true risk of potential cross-reactivity between cephalosporins and penicillins versus the risk of an inadequately treated infection**

The potential risk for allergic cross-reactivity with cephalosporins in a penicillin-allergic patient is estimated at <1% [52]. The risk for cross-reactivity is related to side-chain similarity and not the presence of the beta-lactam ring between the classes. For example, in a patient with a true allergy to amoxicillin, cefadroxil and cephalexin should be avoided because they have an identical side chain. Later-generation cephalosporins or those with different side chains, such as cefazolin, cefuroxime, or cefdinir, would be considered safe to use in this patient.

Data has shown that while 10% of patients encountering healthcare are labeled as penicillin allergic, only 5% of these patients truly have an allergy [53]. These false allergies can drive prescribers to use medications that have a higher cost, may be less effective, cause additional side effects, or contribute to antibiotic resistance. For example, treatment of an uncomplicated urinary tract infection with a fluoroquinolone rather than a cephalosporin increases the risk of renal impairment, tendinitis, and *C. difficile*. It is generally safe to prescribe a cephalosporin to a patient with a non-life-threatening reaction to penicillin.

KEY FACT | **Antibiotics can have significant adverse effects and should only be used when indicated.**

Important adverse reactions

Pitfall | **Failing to consider a drug reaction in patients presenting with rash**

Classic medication reaction patterns include drug-induced exanthems, urticaria (IgE mediated), serum sickness, vasculitis, Stevens-Johnson syndrome (SJS), photosensitivity, and others.

The typical drug-induced exanthem is usually morbilliform (measles-like exanthem with discrete maculopapules) or macular papular. It often begins in dependent areas and then generalizes. It is commonly pruritic and usually occurs within two weeks of taking a new drug or within days of reexposure.

SJS, also known as erythema multiforme major, and toxic epidermal necrolysis are potentially fatal conditions that cause the epidermis to separate from the dermis. Although most cases are idiopathic, the most common known cause is medications. Medications that are known to precipitate SJS include antigout agents (especially allopurinol), antibiotics (sulfonamides, penicillins, cephalosporins, and others), antipsychotics, antiepileptics (including carbamazepine, phenytoin, lamotrigine, phenobarbital), analgesics including NSAIDs, and certain antineoplastic drugs. SJS has a mortality rate of approximately 5%. It must be strongly considered in any patient presenting with new erythematous skin lesions who is taking a medication known to precipitate SJS. In these cases, the suspected medication should be stopped until SJS is ruled out [54]. Patients for whom SJS is suspected should have dermatology consultation rapidly or be transferred to a higher level of care for additional evaluation and potential hospitalization.

Metabolism

Pitfall | Failing to consider metabolism when prescribing medications

Variability of metabolism due to genetic factors or concomitant medications can dramatically alter the effect of an agent on a patient. For example, warfarin is a commonly used vitamin K antagonist that is metabolized by the p450 pathway in the liver. Because this pathway metabolizes many medications, many medications affect warfarin's metabolism (Box 13.3). Some medications increase its metabolism, potentially leading to life-threatening thromboembolic disease. Others inhibit its metabolism, potentially leading to life-threatening bleeding [55]. Particular caution should be taken when prescribing medications to patients on warfarin due to the potential effects on metabolism and risk of hypercoagulability and subsequent severe bleeding [56]. If patients on warfarin are prescribed antibiotics they should have close follow-up with their anticoagulation clinic, primary care team, or pharmacist to assist with any adjustment needed to warfarin dosing in the short term. Recommendations from the American Geriatric Society are that an international normalized ratio (INR) should be monitored more frequently after initiation of an antibiotic for patients on warfarin [57].

Prescribers should avoid use of medications known to alter INR in patients on warfarin whenever possible. Obtain INR levels in patients on warfarin therapy with suspected bleeding, especially if there has been recent addition or subtraction of a medication or herbal supplement [55]. If patients require a medication that may interfere with INR management, it is recommended that the clinic managing the INR be contacted for recommendations regarding warfarin dosing changes and expedited follow-up.

Other medications that pose problematic metabolic entanglements include tramadol and codeine. Both medications have well-established variation in metabolism based on genetic profiles. This variability among patients can increase the risk of toxicity, overdose, and death when codeine or tramadol is prescribed [58]. Tramadol additionally has serotonergic and noradrenergic activity that can react with other medications, increasing the risk of serotonin syndrome [59, 60]. Although tramadol was a schedule IV medication for several years, it has been associated with an increased risk of chronic opioid use [48]. A trend of increased use of tramadol on discharge from emergency departments with an associated decrease in other short-acting opioids [49] may reflect a mindset that tramadol is a somehow less concerning medication. However, the metabolic profile of tramadol is not one that should be regarded as any safer than other short-acting opioids, such as oxycodone. Prescriptions of tramadol or codeine should be undertaken with care.

Box 13.3 Commonly prescribed medications that interact with warfarin

P450 inhibitors: ciprofloxacin
P450 inducers: prednisone (weak)
P450 metabolism: hydrocodone, glucocorticoids, ondansetron, cyclobenzaprine, codeine, tramadol

Increased effect

- Acetaminophen
- Allopurinol
- Aspirin
- Amiodarone
- Cephalosporins
- Cimetidine
- Ciprofloxacin
- Clopidogrel
- Diclofenac
- Erythromycin
- Fluconazole
- Fluoxetine (and other selective serotonin reuptake inhibitors)
- Glucagon
- Influenza virus vaccine
- Metronidazole
- Macrolide antibiotics
- Omeprazole
- Sulfamethoxazole-trimethoprim
- Thyroid hormone

Decreased effect

- Carbamazepine
- Dicloxacillin
- Haloperidol
- Nafcillin
- Oral contraceptives
- Phenobarbital
- Rifampin
- Vitamin K

Medications with a narrow therapeutic index

Pitfall | Failing to consider medications as a cause of vague nonspecific symptoms

Many commonly used medications can be the cause of nonspecific symptoms for which patients will seek care. A careful review of medications should be performed whenever insidious complaints do not have an obvious etiology. Table 13.3 lists a sample of common medications and their adverse effects once they reach toxic levels.

These medications all have a narrow therapeutic index (the ratio of the amount of the agent that causes a degree of therapeutic effect that causes toxicity) [61–64].

When toxicity is suspected, patients on these medications should have their levels checked or should be transferred to a facility with access to toxicology expertise to identify and mitigate the toxicity. Timing of these levels relative to the last dose taken by the patient is essential to avoid falsely high or low values. It is important to realize that for many of these agents serum levels may not correlate with toxicity in cases of chronic overdose. Poison control centers can offer expertise and guidance. The US Poison Help hotline is available 24 hours every day and can be reached at 1-800-222-1222.

KEY FACT | **Medications have the potential for toxicity, which must be considered when evaluating any patient.**

Pitfalls in special populations

Drugs to avoid in the elderly

Pitfall | Failing to assess risks and benefits of high-risk medications in the elderly

The updated Beers Criteria® for potentially inappropriate medication use in the elderly were published in 2023 [57]. There are over 100 medications that have strong evidence to suggest a high risk of adverse events in older adults. Their initiation should be avoided and consideration given to deprescribing [57]. Adverse reactions leading to arrhythmias, sedation, falls, and anticholinergic adverse effects were common reasons for inclusion. The full list can be accessed via the American Association of Geriatrics (www.americangeriatrics.org). Avoidance of all these medications in elderly patients may be impractical and is best addressed by their primary provider. When considering prescribing for older adults, assessment and discussion of the risks and benefits of use of these medications must occur.

Pitfall | Failing to consider fall risk when prescribing medications with psychotropic effects

Specific classes of medications identified on the Beers list include benzodiazepines and psychotropic medications.

Table 13.3 Drugs with a narrow therapeutic index.

Medication/toxic symptoms	Factors contributing to toxicity
Digoxin	Renal impairment
• Nausea/vomiting	Advanced age
• Confusion	Hypoxia
• Lethargy	Myocardial ischemia
• Depression	Hypothyroidism
• Fatigue	Hypokalemia
• Headache	Hypomagnesemia
• Paresthesia	Hypercalcemia
• Weakness	Acidosis
• Scotomata	Uremia
• Palpitations (dysrhythmias)	Hepatitis
Phenytoin	Hypoalbuminemia
• Dizziness	Valproic acid
• Ataxia	NSAIDs
• Tremors	Amiodarone
• Lethargy	SSRIs
• Nausea and vomiting	Ciprofloxacin
• Slurred speech	Ethanol
• Blurred vision	Pregnancy
• Diplopia	Renal impairment
• Confusion	Dehydration
• Hallucinations	Hepatic failure
Lithium	Cyclosporine
• Nausea/vomiting	Calcium channel
• Tremor	Blockers
• Agitation	Antifungal agents
• Muscle weakness	Erythromycin
• Ataxia	Glucocorticoids
• Confusion	Grapefruit juice
• Dysarthria	
• Rigidity	
Tacrolimus	
• Headache	
• Tremor	
• Nausea/vomiting	
• Diarrhea	
• Alopecia	

NSAID, nonsteroidal anti-inflammatory drug; SSRI, selective serotonin reuptake inhibitor.
Data from Sanchez *et al*. [39]; Martinez-Perez *et al*. [40]; Kohut *et al*. [41]; Spurling *et al*. [42].

Diazepam and other benzodiazepines are frequently prescribed for a number of indications including muscle spasm, anxiety, alcohol withdrawal, and insomnia. The sedative effect has a propensity to increase the risk of falls, particularly in the elderly [65]. Among people older than 65 years, 30% fall at least once per year and the result can be devastating. Long-acting benzodiazepines have been associated with increased risk of hip fractures and hypnotics as a class have been found to be associated with falls [66, 67].

Psychotropic medications have also been associated with an increased number of falls. Selective serotonin reuptake inhibitors (SSRIs) are more strongly related to falls than tricyclic antidepressants. The mechanism may be related to the sedative effect or orthostatic hypotension. Antipsychotics have also been associated with falls, possibly due to sedation, extrapyramidal gait disturbance, or anticholinergic visual disturbance. Hypnotics, antidepressants, and benzodiazepines demonstrate a significant association with falls in the elderly, thus alternatives should be used whenever possible.

Drugs contraindicated in pregnancy

Pitfall | **Failing to consider pregnancy status when prescribing medications**

Several medications have teratogenic and/or embryocidal effects. The fetus is at greatest risk of teratogenic effects during the first trimester. Drug administration during pregnancy can potentially cause congenital anatomic malformations, and can also affect social, intellectual, and functional development. Patients' pregnancy status must be considered when prescribing medications, as many patients in their first trimester are unaware they are pregnant. When using agents in pregnancy, one must weigh the therapeutic benefit with the potential risk to the developing fetus [68]. The US Food and Drug Administration (FDA) issues a risk assessment for each medication, but it is still common for there to be insufficient evidence. The prior classification of categories A–D and X has been phased out. New risk assessment categories include fetal risk is minimal, fetal risk cannot be ruled out, may cause fetal harm, fetal harm has been demonstrated, and contraindicated. This grouping can oversimplify a complex topic. We recommend having available a more in-depth text or reference to consult when specific questions arise. Prescribers must be familiar with the risk categories and the meaning behind them when there is sufficient evidence to make a recommendation and when there is not. The degree of the potential adverse effect must be acknowledged, and an assessment of the risks and benefits of medication use in pregnant patients must occur in discussion with the patient if pursuing prescription.

Evidence to support or deter prescription may emerge at any time and prescribers should update their working knowledge of commonly prescribed medications regularly and uncommonly prescribed medications in real time.

Additional care should be taken when using agents that may have embryocidal effects in consideration of recent changes to the medico-legal landscape regarding state-by-state rulings on the legal status of a fetus.

Pitfall | **Failing to adhere to medication alerts when using electronic prescribing, or not using electronic prescribing**

Patient safety and prevention of medication errors are top priorities for healthcare organizations. Prevention of errors at the prescribing stage is an essential component. Prescribing problems leading to patient harm can arise from prescribing medications with potential drug–drug interactions, inappropriate doses, contraindications, allergies, or drug duplication. Failure to warn of potential adverse drug reactions can also have negative consequences. Fortunately, pharmacists often act as a "safety net" and are able to detect problem prescriptions. However, it is unreasonable to believe that pharmacists will catch all problematic prescriptions. When mistakes are identified, corrective action is typically time consuming for the pharmacist and may result in a repeat visit to the prescribing provider.

With widespread use of computerized electronic health records and digital transfer of prescriptions, all practitioners should have ready access to a computerized decision support system to reduce prescribing errors. Benefits of computerized prescribing include speed, improved legibility, and automated record keeping [69]. Decision support with warnings of clinically significant interactions or potential dosing errors is essential for preventing prescribing errors. If computerized prescribing is not available, an electronic support instrument should be used to check for dosing, contraindications, drug interactions, need for monitoring, and anticipatory guidance for adverse effects. If this is not available, consultation with a pharmacist or a drug handbook is advised.

KEY FACT | **Use of electronic prescribing tools can mitigate error and improve patient safety. When they are unavailable, other reference sources should be used to identify potential cross-reactions and dosing safety.**

Pitfall | **Refilling unfamiliar or high-risk medications**

The landscape of prescription medications is constantly evolving, with new medications, additional indications, or boxed warnings being introduced regularly. The FDA provides a podcast on medication safety updates when they are released [70]. In 2022 there were 447 drug safety-related label changes, including 56 boxed warning updates [71]. In light of these rapid changes, it is important to keep abreast of the current status of commonly prescribed medications and to avoid prescribing or refilling prescriptions without adequate familiarity.

Patients using insulin

Insulin comes in several formulations, ranging from long-acting (duration) to rapid-acting and mixed formulations. Long-acting insulin glargine has an onset over six hours post injection and continued hypoglycemic effects for 24 hours, whereas rapid-acting insulin has an initial effect within minutes, a peak effect within 40–60 minutes, and a duration of only 5–7 hours [6]. Ranges of doses based on patient-specific factors and multiple formulations create a milieu of increased risk. Additionally, patients who have more than one formulation of insulin prescribed have a higher likelihood of experiencing a dosing error resulting in a need for emergency care [72]. When prescriptions/refills are necessary to provide in an urgent care setting, close coordination with a pharmacist, review of the patient record, and verification of current doses are highly recommended to ensure safety.

Pearls for improving patient outcomes

- Overall, the risk of cephalosporin-penicillin allergic cross-reactivity is low and decreases with higher-generation cephalosporins; risks and benefits should be assessed on an individual basis.
- Always understand and counsel patients on potential adverse drug events.
- Avoid prescribing nonsteroidal anti-inflammatory drugs to patients at high risk for renal failure or gastrointestinal bleeding.
- Recognize products that contain acetaminophen and include these when determining acetaminophen dosing.
- When using local anesthetics, always calculate safe dosing parameters and minimize pain of injection.
- Identify patients with prolonged QTc intervals and avoid drugs that could cause ventricular arrhythmia.
- Use antibiotics judiciously only when indicated.
- Reference the Beers list when prescribing to older adults.
- Avoid medications that increase falls risk in the elderly.
- Identify drug-related reactions and rashes and discontinue offending agents, referring to a higher level of care when appropriate (e.g. Stevens-Johnson syndrome).
- Recognize drugs with a high risk of interactions and take appropriate monitoring steps to avoid complications.
- When prescribing for pregnant patients, always reference an up-to-date source regarding potential fetal risks.
- Carefully review warnings from electronic prescribing systems and take steps to weigh risks and benefits before clicking through them.
- Avoid prescribing high-risk and unfamiliar medications.

References

1 U.S. Food and Drug Administration. FAERS reporting by patient outcomes by year. 2015. https://www.fda.gov/drugs/questions-and-answers-fdas-adverse-event-reporting-system-faers/faers-reporting-patient-outcomes-year. Accessed July 23, 2024.
2 Xu J, Kochanek KD, Murphy S, *et al*. Deaths: final data for 2007. *Natl Vital Stat Rep* 2010; **58**(19): 1–19.
3 Zed PJ, Abu-Laban RB, Balen RM, *et al*. Incidence, severity and preventability of medication-related visits to the emergency department: a prospective study. *CMAJ* 2008; **178**(12): 1563–1569.
4 Musumba C, Pritchard DM, Pirmohamed M. Review article: cellular and molecular mechanisms of NSAID-induced peptic ulcers. *Aliment Pharmacol Ther* 2009; **30**(6): 517–531.
5 Chang RW, Tompkins DM, Cohn SM. Are NSAIDs safe? Assessing the risk-benefit profile of nonsteroidal anti-inflammatory drug use in postoperative pain management. *Am Surg* 2021; **87**(6): 872–879.

6 Micromedex® Healthcare Series. n.d. https://www.micromedexsolutions.com/home/dispatch. Accessed July 23, 2024.
7 Brent J, Palmer R. Acetaminophen. In: Wolfson AB, Harwood-Nuss A (eds.), *Harwood-Nuss Clinical Practice of Emergency Medicine*. Philadelphia (PA): Lippincott Williams and Wilkins; 2005: pp. 1477–1481.
8 Freo U. Paracetamol for multimodal analgesia. *Pain Manag* 2022; **12**(6): 737–750.
9 Godfrey RG, de la Cruz S. Effect of ibuprofen dosage on patient response in rheumatoid arthritis. *Arthritis Rheum* 1975; **18**(2): 135–137.
10 Simon LS, Mills JA. Nonsteroidal antiinflammatory drugs (second of two parts). *N Engl J Med* 1980; **302**(22): 1237–1243.
11 Lynch ME, Clark AJ, Sawynok J, *et al*. Topical amitriptyline and ketamine in neuropathic pain syndromes: an open-label study. *J Pain* 2005; **6**(10): 644–649.
12 PDQ Supportive and Palliative Care Editorial Board. *PDQ Cancer Information Summaries: Pain (PDQ®)*. Bethesda (MD): PDQ; 2015.
13 Crowley AP, Sun C, Yan XS, *et al*. Disparities in emergency department and urgent care opioid prescribing before and after randomized clinician feedback interventions. *Acad Emerg Med* 2023; **30**(8): 809–818.
14 Deyo RA, Von Korff M, Duhrkoop D. Opioids for low back pain. *BMJ* 2015; **350**: g6380.
15 Groban L. Central nervous system and cardiac effects from long-acting amide local anesthetic toxicity in the intact animal model. *Reg Anesth Pain Med* 2003; **28**(1): 3–11.
16 Mulroy MF, Hejtmanek MR. Prevention of local anesthetic systemic toxicity. *Reg Anesth Pain Med* 2010; **35**(2): 177–180.
17 Todd KH, Ducharme J, Choiniere M, *et al*. Pain in the emergency department: results of the pain and emergency medicine initiative (PEMI) multicenter study. *J Pain* 2007; **8**(6): 460–466.
18 Tran AN, Koo JY. Risk of systemic toxicity with topical lidocaine/prilocaine: a review. *J Drugs Dermatol* 2014; **13**(9): 1118–1122.
19 Oni G, Brown S, Kenkel J. Comparison of five commonly-available, lidocaine-containing topical anesthetics and their effect on serum levels of lidocaine and its metabolite mono-ethylglycinexylidide (MEGX). *Aesthet Surg J* 2012; **32**(4): 495–503.
20 Mills AM, Shofer FS, Boulis AK, *et al*. Racial disparity in analgesic treatment for ED patients with abdominal or back pain. *Am J Emerg Med* 2011; **29**(7): 752–756.
21 Eggleston ST, Lush LW. Understanding allergic reactions to local anesthetics. *Ann Pharmacother* 1996; **30**(7–8): 851–857.
22 Christoph RA, Buchanan L, Begalla K, *et al*. Pain reduction in local anesthetic administration through pH buffering. *Ann Emerg Med* 1988; **17**(2): 117–120.
23 Colaric KB, Overton DT, Moore K. Pain reduction in lidocaine administration through buffering and warming. *Am J Emerg Med* 1998; **16**(4): 353–356.
24 Scarfone RJ, Jasani M, Gracely EJ. Pain of local anesthetics: rate of administration and buffering. *Ann Emerg Med* 1998; **31**(1): 36–40.
25 McGee DL. Local and topical anesthesia. In: Roberts JR (ed.), *Roberts and Hedges Clinical Procedures in Emergency Medicine and Acute Care* (7e edn.). Philadelphia (PA): Elsevier; 2019: pp. 523–544.
26 Konigs I, Wenskus J, Boettcher J, *et al*. Lidocaine-epinephrine-tetracaine gel is more efficient than eutectic mixture of local anesthetics and mepivacaine injection for pain control during skin repair in children: a prospective, propensity score matched two-center study. *Eur J Pediatr Surg* 2020; **30**(6): 512–516.
27 Nair M, Gurunathan D. Comparative evaluation of the efficacy of two anesthetic gels (2% lignocaine and 20% benzocaine) in reducing pain during administration of local anesthesia – a randomized controlled trial. *J Anaesthesiol Clin Pharmacol* 2019; **35**(1): 65–69.
28 Barnes BJ, Hollands JM. Drug-induced arrhythmias. *Crit Care Med* 2010; **38**(6 Suppl): S188–S197.
29 Farzam K, Tivakaran VS. QT prolonging drugs. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2023. https://www.ncbi.nlm.nih.gov/books/NBK534864. Accessed July 23, 2024.
30 Putnam KT, Wilcox M, Robertson-Blackmore E, *et al*. Clinical phenotypes of perinatal depression and time of symptom onset: analysis of data from an international consortium. *Lancet Psychiatry* 2017; **4**(6): 477–485.
31 UpToDate. Some reported causes and potentiators of the long QT syndrome. 2024. https://www.uptodate.com/contents/image?imageKey=DRUG/57431. Accessed July 29, 2024.
32 Charbit B, Alvarez JC, Dasque E, *et al*. Droperidol and ondansetron-induced QT interval prolongation: a clinical drug interaction study. *Anesthesiology* 2008; **109**(2): 206–212.
33 McKechnie K, Froese A. Ventricular tachycardia after ondansetron administration in a child with undiagnosed long QT syndrome. *Can J Anaesth* 2010; **57**(5): 453–457.
34 Cubeddu LX. QT prolongation and fatal arrhythmias: a review of clinical implications and effects of drugs. *Am J Ther* 2003; **10**(6): 452–457.
35 Molassiotis A, Affronti ML, Fleury M, *et al*. 2023 MASCC/ESMO consensus antiemetic guidelines related to integrative and non-pharmacological therapies. *Support Care Cancer* 2023; **32**(1): 30.
36 Erdogan-Ongel E, Heung Y, Rozman de Moraes A, *et al*. Inhalation of isopropyl alcohol for the management of nausea and vomiting: a systematic review. *J Palliat Med* 2023; **26**(1): 94–100.

37 Journal of Urgent Care Medicine. Top 20 medications prescribed. 2016. https://www.jucm.com/top-20-medications-prescribed. Accessed July 23, 2024.

38 Centers for Disease Control. Antibiotic use in the United States, 2022 update: progress and opportunities. 2022. https://archive.cdc.gov/#/details?url=https://www.cdc.gov/antibiotic-use/stewardship-report/2022.html. Accessed July 23, 2024.

39 Sanchez GV, Fleming-Dutra KE, Roberts RM, *et al*. Core elements of outpatient antibiotic stewardship. *MMWR Recomm Rep* 2016; **65**(RR-6): 1–12.

40 Martinez-Perez C, Alvarez-Peregrina C, Villa-Collar C, *et al*. Citation network analysis of the novel coronavirus disease 2019 (COVID-19). *Int J Environ Res Public Health* 2020; **17**(20): 7690.

41 Kohut MR, Keller SC, Linder JA, *et al*. The inconvincible patient: how clinicians perceive demand for antibiotics in the outpatient setting. *Fam Pract* 2020; **37**(2): 276–282.

42 Spurling GK, Del Mar CB, Dooley L, *et al*. Delayed antibiotics for symptoms and complications of respiratory infections. *Cochrane Database Syst Rev* 2017; **9**(9): CD004417.

43 Keller SC, Tamma PD, Cosgrove SE, *et al*. Ambulatory antibiotic stewardship through a human factors engineering approach: a systematic review. *J Am Board Fam Med* 2018; **31**(3): 417.

44 Little P, Stuart B, Francis N, *et al*. Antibiotic prescribing for acute respiratory tract infections 12 months after communication and CRP training: a randomized trial. *Ann Fam Med* 2019; **17**(2): 125–132.

45 Havers FP, Hicks LA, Chung JR, *et al*. Outpatient antibiotic prescribing for acute respiratory infections during influenza seasons. *JAMA Netw Open* 2018; **1**(2): e180243.

46 Shulman ST, Bisno AL, Clegg HW, *et al*. Clinical practice guideline for the diagnosis and management of group a streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. *Clin Infect Dis* 2012; **55**(10): 1279–1282.

47 Boggs KM, Glew D, Rahman KN, *et al*. Pediatric telehealth use in U.S. emergency departments in 2019. *Telemed J E Health* 2023; **29**(4): 551–559.

48 Thiels CA, Habermann EB, Hooten WM, *et al*. Chronic use of tramadol after acute pain episode: cohort study. *BMJ* 2019; **365**: l1849.

49 Mullins N, Kang JE, Campos AI, *et al*. Dissecting the shared genetic architecture of suicide attempt, psychiatric disorders, and known risk factors. *Biol Psychiatry* 2022; **91**(3): 313–327.

50 Kutty PK, Woods CW, Sena AC, *et al*. Risk factors for and estimated incidence of community-associated Clostridium difficile infection, North Carolina, USA. *Emerg Infect Dis* 2010; **16**(2): 197–204.

51 Centers for Disease Control. Antibiotic resistance threats in the United States 2019. 2019. https://www.cdc.gov/drugresistance/pdf/threats-report/2019-ar-threats-report-508.pdf. Accessed July 23, 2024.

52 Exius R, Gabrielli S, Abrams EM, *et al*. Establishing amoxicillin allergy in children through direct graded oral challenge (GOC): evaluating risk factors for positive challenges, safety, and risk of cross-reactivity to cephalosporines. *J Allergy Clin Immunol Pract* 2021; **9**(11): 4060–4066.

53 Liu MY, McCoul ED, Brooks EG, *et al*. Inaccurate penicillin allergy labels: consequences, solutions, and opportunities for rhinologists. *Int Forum Allergy Rhinol* 2023; **13**(6): 973–978.

54 Hazin R, Abuzetun JY, Khatri KA. Derm diagnoses you can't afford to miss. *J Fam Pract* 2009; **58**(6): 298–306.

55 Hogg K, Weitz JI. Blood coagulation and anticoagulant, fibrinolytic, and antiplatelet drugs. In: Brunton LL, Hilal-Dandan R, Knollmann BC (eds.), *Goodman & Gilmans: The Pharmacological Basis of Therapeutics* (13e edn.). New York: McGraw Hill Education; 2017: ch. 32.

56 Baillargeon J, Holmes HM, Lin Y-L, *et al*. Concurrent use of warfarin and antibiotics and the risk of bleeding in older adults. *Am J Med* 2012; **125**(2): 183–189.

57 By the 2023 American Geriatrics Society Beers Criteria® Update Expert Panel. American Geriatrics Society 2023 updated AGS Beers criteria® for potentially inappropriate medication use in older adults. *J Am Geriatr Soc* 2023; **71**(7): 2052–2081.

58 Aly SM, Hakim F, Richeval C, *et al*. Metabolic ratios and SNPs implicated in tramadol-related deaths. *Int J Legal Med* 2023; **137**(5): 1431–1437.

59 Bianconi G, Malissin I, Labat L, *et al*. Marked and prolonged serotonin toxicity in a tramadol-poisoned patient with a pharmacokinetic study. *Clin Toxicol (Phila)* 2022; **60**(3): 382–385.

60 Puszkiel A, Malissin I, Cisternino S, *et al*. Massive tramadol ingestion resulting in fatal brain injury – a pharmacokinetic study with discussion on the involved mechanisms of toxicity. *Clin Toxicol (Phila)* 2022; **60**(9): 1059–1062.

61 Kashani JS, Gerkin RD. Cardiac glycosides. In: Wolfson AB (ed.), *Harwood-Nuss Clinical Practice of Emergency Medicine*. Philadelphia (PA): Lippincott Williams and Wilkins; 2005: pp. 1570–1572.

62 Kalimullah EA, Aks SE. Phenytoin. In: Wolfson AB (ed.), *Harwood-Nuss Clinical Practice of Emergency Medicine* (7e edn.). Philadelphia, PA: Lippincott, Williams and Wilkins; 2015: pp. 1413–1416.

63 Kulig KW. Lithium. In: Wolfson AB (ed.), *Harwood-Nuss Clinical Practice of Emergency Medicine*. Philadelphia (PA): Lippincott, Williams and Wilkins; 2005: pp. 1625–1626.

64 Krensky AM, Bennett WM, Vincenti F. Immunosuppressants, tolerogens, and immunostimulants. In: Brunton L, Chabner BA, Knollmann BC (eds.), *Goodman & Gilmans The Pharmacological Basis of Therapeutics*. New York: McGraw Hill; 2011: pp. 1005–1029.

65 Andrade C. Sedative hypnotics and the risk of falls and fractures in the elderly. *J Clin Psychiatry* 2018; **79**(3): 8f12340.
66 Cumming RG. Epidemiology of medication-related falls and fractures in the elderly. *Drugs Aging* 1998; **12**(1): 43–53.
67 Woolcott JC, Richardson KJ, Wiens MO, *et al*. Meta-analysis of the impact of 9 medication classes on falls in elderly persons. *Arch Intern Med* 2009; **169**(21): 1952–1960.
68 Briggs GG, Freeman RK, Yaffe SJ. *Drugs in Pregnancy and Lactation: A Reference Guide to Fetal and Neonatal Risk*. Philadelphia (PA): Lippincott, Williams and Wilkins; 2005.
69 Chen YF, Neil KE, Avery AJ, *et al*. Prescribing errors and other problems reported by community pharmacists. *Ther Clin Risk Manag* 2005; **1**(4): 333–342.
70 U.S. Food & Drug Administration. FDA drug safety podcasts. n.d. https://www.fda.gov/drugs/drug-safety-and-availability/fda-drug-safety-podcasts. Accessed July 23, 2024.
71 U.S. Food & Drug Administration. Drug safety-related labeling changes (SrLC). 2023. https://www.accessdata.fda.gov/scripts/cder/safetylabelingchanges/index.cfm?event=searchResult.page. Accessed July 23, 2024.
72 Geller AI, Conrad AO, Weidle NJ, *et al*. Outpatient insulin-related adverse events due to mix-up errors: findings from two national surveillance systems, United States, 2012–2017. *Pharmacoepidemiol Drug Saf* 2021; **30**(5): 573–581.

CHAPTER 14
Talking the Talk: Effective Communication in Urgent Care

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Introduction

Urgent care takes place in settings of constant communication. As acute care providers we take histories, answer phones, explain options, recommend options, and arrange for follow-up. While doing this we are frequently interrupted, often asked for clarification, and occasionally misunderstood [1]. Interruptions provide a nice example of the toll that normal communications may take on clinical activity and the complexity of the topic. Interruptions in emergency care are frequent and our return to the interrupted task is not guaranteed [2, 3]. Sometimes an interruption may be necessary to assure the safety of the patient, as when a new result suggests a diagnosis more severe than initially considered; more often an interruption may lead to distraction, disorganization, and delay [4]. The simple solution, to make a "no interruption" rule, would not provide a satisfactory result. The obvious corollary, "interrupt only when important and necessary," is too vague to put into effect. In clinical practice a keen appreciation of the pitfall may be our best approach: "Pitfall: Underappreciating the cognitive danger of interruptions."

The enormity of the field of communications precludes a comprehensive review in a single chapter. Therefore, what follows are common pitfalls that may appear any day, at any time, in urgent care. They are arranged in the order of a typical visit: introduction and history taking, evaluation, testing and results, discharge, and follow-up. Because communication is inherently social, the pitfalls and associated suggestions must be considered within the cultural dynamic of the department. This does not mean that the specific pitfalls do not exist everywhere; most of them do. It does mean that their understanding and interpretation should be adjusted to local conditions and norms.

Pitfall | **Thinking that communication does not matter**

If you have made it this far in this book, it is unlikely that we need to convince you of the importance of good communication. Just in case, it is worth considering why we bother discussing communication, much less researching it.

From a very basic point of view, "communication failure" frequently appears in the list of what went wrong during the evaluation of a medical event gone awry. This may be on account of the density of communication in clinical practice and points out the room for improvement [5]. Training in specific communication skills has been shown to improve the medical student's ability to organize, manage time, negotiate, and share decision making, all potentially improving patient relations and patient outcomes [6]. Our ability to communicate, and an appreciation of its subtleties, can improve relations both with patients and with staff, making for better patient outcomes and a more satisfying

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career [7, 8]. Effective communication may improve patient satisfaction, bolster relationships with other providers, and prevent litigation [9, 10].

The concept that the provider speaks and the patient hears and understands is too convenient, and simply wrong. Communication demands both speaking and listening—and confirming that what has been said has also been heard [11]. No one likes to think that the day's effort has been lost to misunderstanding. By reducing conflict and improving relations effective communication leads to a greater sense of accomplishment and overall job satisfaction.

KEY FACT | **Effective communication may improve patient satisfaction, bolster relationships with other providers, and prevent litigation.**

Pitfall | **Ignoring the "little" things**

Dale Carnegie provided a highly memorable way of expressing that the "little" things matter. He asks that we "remember that a person's name is to that person the sweetest and most important sound in any language" [12]. The rule holds in urgent care as in the rest of medicine. While the nature of the greeting may change according to local culture, a correct and respectful means of address puts the patient encounter along the right path. This is, incidentally, also a safety mechanism. A proper introduction helps confirm the correct room and patient.

What are the other little things that appear to make a difference? Sitting down when talking with the patient increases the perceived length of the visit [13], suggesting that the time taken to find a chair or stool is well spent. Saying hello, shaking hands, and confirming who is in the room start the relationship on an effective trajectory, demonstrate an understanding that an urgent care visit is both a medical and a social event, provide an opportunity to make an initial assessment of the clinical situation, and help to make sure that no one in the room will be hearing something the patient would prefer that they did not. Apologizing for inconvenience up front, especially if running late or if something else has already happened to upset the patient, can prevent the need for subsequent explanation and potentially hours of follow-up activity. A quick apology for a little inconvenience may keep it from growing into a larger headache. Remembering good manners goes a long way toward getting the little things right [13]. Good manners also help everyone remember that the patient is at the center of the visit.

KEY FACT | **Sitting down when talking with the patient increases the perceived length of the visit.**

Pitfall | **Failing to understand why the patient has come**

Much of what is seen in urgent care is self-limited illness and much of what we provide is reassurance. Patients do not necessarily have to come for an urgent care visit—though they may not appreciate this—and, in general, they make a choice to be seen. Typically there is a compelling reason why a patient has chosen to seek urgent care. And there is always a tradeoff in their decision to visit: They may have to spend money to be seen; they may lose time from work; they may simply lose time from their day.

The compelling reason for the visit may not be immediately evident, particularly if we ignore the social aspects of illness. Much of the reason for the visit may be medical, such as worsening pain or a cough that has lasted longer than the patient thinks it should, and much of the reason may be social, such as the need for a work note. Patients may want a specific medication, a particular approach, or satisfaction for a family member or friend who has pushed them to seek care. If the underlying reason for the visit is not understood, the patient may depart unsatisfied and the care provider may be mystified. Often patients will shroud a social reason for a visit within a medical context. If the care provider does not ask about the social context, the opportunity for accurate communication may be lost. For this reason, it is essential to ask about family, job, and social needs. A few questions may prove helpful:

- What made you change your plans today to come for a visit?
- What can I do for you that would be most helpful?
- What are you hoping I can do for you today?
- Is there something different going on at home? At work?
- Is there anything else you need today?
- Is there anything more that I can do for you today?

KEY FACT | **The secret in caring *for* the patient is caring *about* the patient.**

To the degree that people have a choice over when to seek care, urgent care must satisfy the patient that their needs have been met. From this perspective, urgent care is fundamentally about patient satisfaction. Furthermore, evidence suggests that the happier the patient is with the treatment, the more positively they will view the treatment, and the better they will feel. Where symptoms may be subjective, including the degree of pain from a sprain and the inconvenience of a runny nose, essential aspects of the treatment are encouragement and explanation [14]. It is essential to understand why the patient has come. This does not mean that we must give *exactly* that. It does mean that we need to recognize the desire, respond appropriately, and potentially negotiate an alternative. In the words of Francis W. Peabody, "the secret of the care of the patient is in caring for the patient" [15].

Pitfall | **Ignoring local news and events**

One of the defining features of urgent care is that patients decide when they seek care. Their determination of their own potential illness may be strongly influenced, however, by family and friends—and by the media. Movies, shows, and social media all convey news of recent illness among celebrities, recent events, and nascent medical theories that can convince patients of the need for evaluation. Celebrities frequently use this to widen knowledge of certain illnesses, so we are more aware of lupus from Salena Gomez and Parkinson's disease from Michael J. Fox.

Local news and events can be equally influential. An outbreak of illness may convince people of the need to see a physician even when they are urged to stay home if the symptoms are not severe. The heterogeneity of COVID-19 incidence and vaccination penetrance provides an obvious example of local variability and a clear event in which appreciation for local understanding and interpretation of events informed the approach to care. Any infectious disease may cause a local response. The rumor of a case of meningococcal meningitis may lead to an influx of exposed individuals concerned that they need a prophylaxis. A local case of rabies is likely, for a time, to increase the number of patients with dog and cat bites who present for acute care.

The appearance of local physicians and hospitals in the news may also influence patients' decisions to seek evaluation. If a well-known physician or hospital appears in the news for having provided anything less than excellent care, concerned patients, having lost their trust in the person or institution from which they would normally seek care, may seek second or third opinions, including from urgent care.

Patients may not recognize or express the role of local events in guiding them to seek care. Only with careful observation, and with an eye toward local news and events, will the astute acute caretaker put it all together. A few questions regarding local news may put the patient at ease and assure patient and caretaker that symptoms have not changed as much as their interpretation in light of recent events. A shared understanding of circumstance both makes the diagnosis and helps communicate its significance.

Pitfall | **Pretending money is not part of the medical relationship**

In determining to seek urgent care, the patient first decides whether the costs will be worth the potential benefit received. The costs take many forms, including time spent, effort in transportation, explanation to friends and family, and, not to be ignored, financial charges. How costs are calculated depends on multiple factors including the patient's insurance, the structure of the medical system, and the nature of the urgent care. Some urgent care centers are quite clear, posting charges on a sign at the entrance; others hide the costs until a bill arrives later.

No matter the arrangement, the costs of medical care are loci for frustration and often best handled up front. (From this point we are considering primarily financial cost, so the exception may be those systems in which the patient sees few or no charges.) Patient complaints typically occur when patients later receive a bill, particularly if they find the charges unexpected or out of line with the services they feel they received. Ignoring these costs, and pretending them away during the visit, only increases the risk of dissatisfaction.

Costs of therapy may also be too easily ignored. Physical therapy may be suggested for back pain or complex drug regimens may be written for a person with asthma, but if the patient cannot afford the physical therapy or the medications, the treatments are of little use. With some straightforward discussion that recognizes the limitations an alternative approach may be found, for example self-directed exercises or alternative pharmaceuticals.

Pitfall | **Failing to explain to the patient what you are not doing—and why you are not doing it**

Urgent care embraces the evaluation of common problems. This brings with it a specific challenge: Many patients have experienced the same problem before, or have friends or family members who have. If not, then they may have researched the problem and potential solutions using both medical sites and social media. As a result, they come to urgent care with preformed expectations for evaluation and care. If these expectations match what is done, then the patient is likely to be satisfied; if not, it is a short path to a dissatisfied patient who is all too willing to place the wisdom of friends, neighbors, and family above that of the acute care clinician.

If the patient asks a question or otherwise indicates what is expected, this becomes much less of a problem. But many patients will not tell. They will simply leave dissatisfied. It is our job to predict what patients may want and either do it, offer it, or explain why we are not doing it. The decision of which of the three approaches to take depends on the patient, location, and practice style. For example, the middle-aged patient with one week of low back pain after exercising for the first time in a year may anticipate or even demand magnetic resonance imaging (MRI) of the spine. Some acute care clinicians may agree to such a request. Others may explain to the patient why this is not necessary, then offer the study as an alternative approach. Still others may reject the request and explain why.

Regardless of the approach, many of these expectations are predictable. By way of illustration, Table 14.1 lays out some of the more common clinical conditions, associated studies requested, and discussion points that help to explain why the studies may not be necessary. This discussion should not imply that the interventions are useless and should always be avoided, but rather that they can be avoided much of the time. When they are avoided, good communication includes explaining to the patient why the study or intervention was not necessary.

KEY FACT | **Patients come to the urgent care with preformed expectations for evaluation and care.**

How does this play out in clinical practice? Consider the case started earlier: the 40-year-old male who has low back pain of one week's duration, worse with movement, better with rest, and without any neurologic findings on examination. Before the patient asks, the acute care clinician explains: "Our goal in this case is to make you feel better. Many people wonder about x-rays after a week of low back pain. While we *could* get them if you truly want, in your case I'm happy to say that they are not necessary. In general, for pain like yours they take time, waste money and resources, and expose you to radiation that you don't need. They would show us broken bones, but we already know you don't have any broken bones. I recommend against them. Most of the time back pain gets better on its own and we'll give yours the benefit of time. To help you over that time, we'll work on some things to make you feel better. If the pain persists for more than a month, then we can reconsider the x-rays."

There are a few additional points worth making about this conversation. The acute care clinician has removed

Table 14.1 Clinical conditions and low-yield interventions that patients may want or expect.

Chief complaint	Studies or treatments that may require explanation	Discussion points
Headache	CT of the brain	Cost, radiation exposure, low yield
	MRI of the brain	Cost, false-positive potential, availability
Back pain	Lumbar spine x-rays	Time, radiation exposure, low yield
	MRI of the spin	Cost, false-positive potential, availability, low yield
Sore throat	Antibiotics	Side effects, lack of benefit, cost
Otitis media	Antibiotics	Side effects, lack of benefit, cost
Abdominal pain	CT of the abdomen	Cost, lack of specific diagnosis, time, radiation exposure, contrast reactions
Cold/upper respiratory infection	Antibiotics	Side effects, lack of benefit, cost

CT, computed tomography; MRI, magnetic resonance imaging.

the element of being a gatekeeper from the conversation by offering the test then explaining why it is unnecessary. This removes from the patient's mind the idea that the provider is simply refusing. In addition, the acute care clinician has offered an explanation of when the study might be necessary—after more time has passed—if the pain persists. The clinician has promised to treat the symptoms that prompted the visit. Finally, the clinician has externalized the medical thought process, freeing the patient from having to guess what the clinician might be thinking.

Even the basic language of diagnosis may change the patient's perception of necessary testing. A diagnosis of "low back pain with associated nerve pain" may not inspire a desire for radiography, while a diagnosis of "sciatica" may. In a family practice, patients were more satisfied with care that omitted antibiotic therapy when the diagnosis was "chest cold" rather than "bronchitis" [16]. The term bronchitis seemed to imply that antibiotics were necessary. We may benefit by testing the name of the diagnosis against the planned course of therapy and reconsidering terms if the name implies the need for unnecessary further testing or therapy.

Pitfall | **Keeping secret when something goes awry**

In urgent care practice major errors are likely to be relatively rare. Despite this, mistakes do occur: diagnoses are incorrect, results are sent on the wrong patient, an x-ray finding is missed, follow-up is misplaced, and so on. In these cases the exemplary approach is to let the patient know what went wrong. It can be challenging to discuss with a patient that there has been an error in care [17]. Research suggests, however, that honesty about errors is what patients want, improves relationships, and reduces legal costs [18]. It also forces clinicians and systems to engage in self-reflection and self-improvement, resulting in improvements in future patient care and more confident practice [19]. Medical ethics demands this type of honesty with the patient.

KEY FACT | **Honesty about errors improves relationships and reduces legal costs.**

Pitfall | **Criticizing prior care**

We can be our own worst enemies. In criticizing another clinician, we open ourselves to equivalent criticism. Effectively, we tell the patient that we do not trust each other, so why should the patient trust us. We also create an environment in which we all are open to senseless criticism and so increase the potential for a lawsuit.

Ironically, we are at times critical without even realizing it. We tend to forget that the prior clinician saw the patient at a different stage of illness, likely before symptoms were evident and potentially before the patient was ready to reveal the nature or degree of the symptoms. The diagnosis is always easiest after it is made; it is rarely so straightforward beforehand.

It is certainly permissible and appropriate to reevaluate a prior diagnosis. It is also reasonable to bring the patient in on these thoughts, typically with an expression of understanding that the picture of illness may have evolved since the last evaluation. Statements such as "I understand why they thought this before…" or "I can see how it looked different last time around" can go a long way toward expanding trust and reinforcing a strong patient–clinician relationship. The goal is always the same: to take the best possible care of the patient through all stages of the illness. Understanding that the course of illness may have changed since the prior evaluation also reminds us that it may evolve again. Avoiding thoughtless criticism inspires a humble attitude that communicates to the patient that symptoms and diagnoses may change, and that the clinician has the capacity to review prior decisions and recast recommendations for new circumstances.

There is another obvious approach when wondering what happened previously: contacting the prior clinician. This is of particular importance if that clinician is a regular caregiver for the patient. The urgent care role is, by definition, a temporary one. If there are providers who are regularly involved, continuing care of the patient and professional relations both dictate contacting the other provider when possible [20]. There are often good reasons for what has come before, and the patient is best supported when these reasons are available to all clinicians and considered in subsequent care.

Pitfall | **Underappreciating the benefits and challenges of humor**

Humor in a clinical setting can inspire rapport, confirm a human link between patient and clinician, and move a stressful situation where anxiety limits communication toward a more relaxed event where patient and clinician speak comfortably. It can also misfire, such as

when a comment is interpreted as an insult or a laugh as a sign of not caring. Culture, language, and personal experience all play a role in determining whether humor can be part of a patient relationship. Omitting humor completely from the clinical environment seems soulless; including it inappropriately may be worse.

Where does this leave the individual acute care clinician with regard to humor? For those whose humor is not readily understood, perhaps the approach is to omit any purposeful attempt to be funny or make patients laugh. Occasions will arise, nonetheless, where a comment leads to a laugh and a better interaction; take note of these moments and consider whether they may be generalized. For those convinced that they are funny, even comedians, pause to consider the effects of comments on patient interactions and whether the humor has gone awry, potentially necessitating more explanation to dig out from underneath a joke gone bad. Humor that aids the patient in understanding and coping with the acute event is to be encouraged; humor that intrudes is best avoided.

Pitfall | **Assuming that the patient understood what was said**

We often speak with the assumption that we are being heard [21]. This is true in all aspects of life, and it is an ironic assumption in an acute care medical setting where pain and anxiety make it less likely that we will be understood. The details of prescriptions and discharge instructions can be lost in the confusion of a discharge process. Different languages, different backgrounds, and differing medical literacy work together to exacerbate the problem [22].

The first step to avoid this pitfall is to recognize the potential for misunderstanding. Essential elements of care should both be spoken and written. Understanding can be confirmed by asking the patient to repeat back, in their own words, what was said. A question may also lead to confirmation of understanding, for example: "Do you mind repeating back how you are going to take this medicine? That will help me be sure I explained it correctly." Jargon should be kept to a minimum. Patients and clinicians alike may prefer medical words for the veneer of knowledge that they provide; patients, however, may not understand them. It is the clinician's duty to translate medical language into lay terms for the benefit of the patient without first being asked to do so.

Pitfall | **Failing to provide anticipatory guidance**

Pediatricians are great at anticipatory guidance; they spend years learning to predict children's futures and letting parents know what will happen and when. The rest of us can learn from them. In adult and acute care we also tend to know what will happen in the future, but we are not always consistent in explaining the probable course of events to patients. For example, we know that a typical viral upper respiratory infection will last for two to three weeks. Yet we often give patients the impression that their illness will be better in just a few days, even asking them to return if it lasts longer, perhaps even giving antibiotics if they continue to cough beyond a shorter period of time. The reality is that the viral symptoms are unlikely to end within a few days. In suggesting otherwise, we undermine our own care. If the patient believes that the cold will resolve in a few days and finds instead that the cough has persisted for two weeks, a natural response may be dissatisfaction with the original care and questioning the skill of the clinician. The patient may also wonder about the degree to which the illness has diverged from its expected course and become concerned that something more serious is emerging—that perhaps the cough signifies pneumonia or cancer.

Patients who have experienced a minor motor vehicle collision or other minor musculoskeletal injury are in a similar predicament. If they come in on day 1, we can tell them with confidence that they will likely have more muscle aches on day 2 and probably on day 3; only after the first few days have passed will they begin to feel more comfortable and this improvement will likely progress slowly. Providing information on the expected future course of care allows the patient to face soreness with confidence, potentially preventing a future visit and burnishing the clinician's reputation for wisdom.

Pitfall | **Failing to let people know that things could get worse—and when they should come back**

The trickiest challenge in the provision of urgent care may be recognizing the small number of patients who have a serious illness presenting subtly. While perhaps unlikely, the backache may be cancer, the headache a hemorrhage, and the dizziness a heart attack.

The best ally in uncovering the sicker patients is time. It is essential that we let patients know this and invite

them to take part in the continuing evaluation of their illness by advising them what to look for. If they have a cellulitis ("skin infection" might be the word to use with the patient), they should know to look for initial improvement after 24 hours, and to come back if it is worse after 48 hours or if they develop a fever. Another technique is to draw a black line around the erythematous area and note that redness may extend beyond this in the first 24 hours, but that by 48 hours it should recede behind the drawn border.

At the time of discharge, every patient should receive a set of warning signs suggesting when it is time to return "right away" [23]. This is the corollary to providing anticipatory guidance: We want to let patients know what is to be expected and what is not. These "times to worry" should be written in simple language and featured prominently in discharge instructions.

KEY FACT | **At discharge every patient should receive warning signs of when to return right away.**

Pitfall | Forgetting to let people know you are going the extra mile

Most people visiting urgent care do not know the normal course of evaluation. The only way they know they have received exceptional care is if we tell them. We should let them know what we have done on their behalf. If their regular doctor has been called, let them know. If an unusual study has been obtained—say, a necessary MRI—let them know that this is typically a hard test to obtain and it was important enough for the extra effort necessary to arrange it. For the most part, if we have not told them that we have gone the extra mile, they have no way of knowing.

Pitfall | Imagining the visit ends when the patient leaves

The patient's illness does not end at the time of discharge; neither does our responsibility. Follow-up is essential, particularly for new results or findings, such as may happen when a radiologist reads the study later or a laboratory test requires more time than the visit. The patient should be warned that future results may come to light and should know how these results will be communicated. Requesting a phone number, email address, or street address is an essential part of any visit because it allows for communication of new results.

For the acute care clinician it is equally important to be able to reconnect with the patient if something led to further thoughts later in the evening, or perhaps even led to worriedly staying awake at night. For the benefit of one's own education and one's career satisfaction, it is essential to be able to follow up with that patient. There is little as satisfying in clinical medicine as finding out more of the story. Episodic views of illness are the rule in urgent care; only by following up can we obtain a longer and broader view.

We have several methods readily available to us to continue care and communication after the time of discharge. The most basic of these are discharge instructions that provide both education and guidance as well as plans for follow-up care [24]. A business card handed to the patient at the end of the encounter indicates that the provider does not view the responsibility as over; rather, it is an invitation to follow up. A handwritten note on the back provides an additional touch that illustrates importance. A follow-up phone call or visit is the most labor intensive and also the most careful of follow-up plans. Letting the patient know that they may receive a follow-up call or letter underscores that the relationship continues.

Pearls for improving patient outcomes

- Pay attention to the simple things—say hello, introduce yourself, sit down, and listen.
- Clarify and respond to the patient's chief complaint.
- Pay attention to local news and events to learn what patients know and may worry about.
- Explain to the patient the tests and treatments you are not recommending.
- Confirm that the patient understood what was said.
- Provide anticipatory guidance and let the patient know what indicates that the illness is more severe than originally determined.
- Let the patient and family know when you have gone the extra mile.
- Provide discharge information that includes potential diagnoses, detailed treatment plans, instructions for follow-up, and information on when to return.

References

1 Eisenberg EM, Murphy AG, Sutcliffe K, *et al*. Communication in emergency medicine: implications for patient safety. *Communication Monographs* 2005; **72**: 390–413.

2 Westbrook J, Coiera E, Dunsmuir TM, *et al*. The impact of interruptions on clinical task completion. *Qual Saf Health Care* 2010; **19**: 284–289.

3 Fong A, Ratwani RM. Understanding emergency medicine physicians multitasking behaviors around interruptions. *Acad Emerg Med* 2018; **25**: 1178–1180.

4 Rivera-Rodriguez AJ, Karsh BT. Interruptions and distractions in healthcare: review and reappraisal. *Qual Saf Health Care* 2010; **19**: 304–312.

5 Patterson ES, Wears RL. Beyond "communication failure." *Ann Emerg Med.* 2009; **53**: 711–712.

6 Ydedidia MJ, Gillespie CC, Kachur E. Effect of communication training on medical student performance. *JAMA* 2003; **290**: 1157–1165.

7 Murphy AG, Eisenberg EM, Sutcliffe KM, *et al*. The patient in 4: framing and sense-making in emergency medicine. *Emergency Medicine News*; **25**: 50–53, 55. doi: https://doi.org/10.1097/00132981-200310000-00037.

8 Cameron KA, Engel KG, DM MC, *et al*. Examining emergency department communication through a staff-based participatory research method: identifying barriers and solutions to meaningful change. *Ann Emerg Med* 2010; **56**: 614–622.

9 Stelfox HT, Gandhi TK, Orav EJ, *et al*. The relation of patient satisfaction with complaints against physicians and malpractice lawsuits. *Am J Med* 2005; **118**: 1126–1133.

10 Amick AE, Schrepel C, Bann M, *et al*. From battles to burnout: investigating the role of interphysician conflict in physician burnout. *Acad Med.* 2023; **98**(9): 1076–1082. https://doi.org/10.1097/ACM.0000000000005226.

11 Capella JN, Street RL. Delivering effective messages in the patient-clinician encounter. *JAMA* 2024; **331**(9): 792–793. https://doi.org/10.1001/jama.2024.0371.

12 Carnegie D. *How to Win Friends & Influence People*. New York: Pocket Books; 1936.

13 Berwick DM. What "patient-centered" should mean: confessions of an extremist. *Health Affairs* 2009; **28**: w555–w565.

14 Colloca L, Barsky AJ. Plaebo and nocebo effects. *N Engl J Med* 2020; **382**: 554–561. doi: 10.1056/NEJMra1907805.

15 Dieppe P, Fussell I, Warber SL. The power of caring in clinical encounters. *BMJ* 2020; **371**: m4100. https://doi.org/10.1136/bmj.m4100.

16 Phillips TG, Hickner J. Calling acute bronchitis a chest cold may improve patient satisfaction with appropriate antibiotic use. *J Am Board Fam Pract* 2005; **18**(6): 459–463.

17 Gallagher TH, Garbutt JM, Waterman AD, *et al*. Choosing your words carefully: how physicians would disclose harmful medical errors to patients. *Arch Intern Med* 2006; **166**: 1585–1593.

18 Witman A, Park D, Hardin S. How do patients want physicians to handle mistakes? A survey of internal medicine patients in an academic setting. *Arch Intern Med* 1996; **156**: 2565–2569.

19 Wears RL, Wu AW. Dealing with failure: the aftermath of errors and adverse events. *Ann Emerg Med* 2002; **39**: 344–346.

20 Stillman MD. Physicians behaving badly. *JAMA* 2008; **300**: 21–22.

21 Makaryus AN, Friedman EA. Patients' understanding of their treatment plans and diagnosis at discharge. *Mayo Clin Proc* 2005; **80**: 991–994.

22 Clarke C, Friedman SM, Shi K, *et al*. Emergency department discharge instructions comprehension and compliance study. *Can J Emerg Med* 2005; **7**: 5–11.

23 Yu KT, Green RA. Critical aspects of emergency department documentation and communication. *Emerg Med Clin North Am* 2009; **27**: 641–654.

24 Taylor DM, Cameron PA. Discharge instructions for emergency department patients: what should we provide? *J Accid Emerg Med* 2000; **17**: 86–90.

CHAPTER 15
Medical Malpractice in Urgent Care: Picking the Low-Hanging Fruit

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The case

A 23-year-old woman who uses oral contraceptives presents to urgent care with four days of sharp and intermittent chest pain, which is nonexertional, nonradiating, worse with range of motion, and without dyspnea or diaphoresis. She has a six-day history of rhinorrhea and a productive cough with yellow sputum, which became blood tinged this morning after a particularly vigorous episode of coughing. She denies pain or swelling of the lower extremities, recent surgery, or history of a thromboembolic disorder. Her chest pain is worse with palpation. Otherwise her vital signs and physical exam are normal. If a pulmonary embolism is later diagnosed, was this malpractice?

The discussion

Every day a fine balancing act takes place across the United States, with practices differing in terms of data gathering, the differential diagnosis, and disposition decisions based on the clinician's risk tolerance [1, 2]. It would be foolish to discount the bedside clinician's legal concern for a misdiagnosis, and it is tricky to estimate how to balance the risk of serious disease with concern for our own careers [3]. We are notoriously poor at estimating patients' expectations [4], so altering our normal best practices to meet our *perception of a patient's expectations* might even cause harm. One study showed that patients with the highest satisfaction have higher healthcare expenditures and a higher mortality rate [5].

Consider being in the urgent care center faced with the scenario outlined above. This is a common occurrence at the bedside: "When should we order a test, refer the patient to a specialist, or send the patient to the emergency department (ED)?" The answer is quite simple in many scenarios: If the risk of the intervention (or test) is lower than the risk of missing the disease, we should proceed with the intervention (or test).

There are some additional factors playing into the decision-making process, including the concerns of the patient or their family, the availability of tests and timeliness of results, the potential for false-positive/incidental findings, as well as the tolerance and understanding of risk by both the patient and the clinician [6]. However, the patient's health should be the ultimate focus guiding this decision-making process.

Medical malpractice risk

The good news is that the average clinician has a very low risk of seeing the inside of a courtroom. Even if they do, the chance of a defense verdict is high. Wong *et al*. looked at 6,770 closed claims from the ED and urgent care, finding that almost two-thirds of cases were dropped, withdrawn, or dismissed [7]. Only 7.6% of cases went to trial, and of

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these almost 93% of juries returned a verdict for the defendant. The most common medical error was failure to diagnose. The top five outcomes cited in these closed claims were (1) cardiac or respiratory arrest, (2) acute myocardial infarction, (3) aortic aneurysm, (4) pulmonary embolism, and (5) appendicitis.

KEY FACT | **The most common factor in medical malpractice cases is "failure to diagnose."**

The top five conditions in malpractice cases with pediatric patients were (1) cardiac or respiratory arrest, (2) appendicitis, (3) disorders of male genital organs, (4) encephalopathy, and (5) meningitis. The chief factor cited was error in diagnosis in 42% of closed claims and 51% of paid claims [8].

KEY FACT | **The top five conditions present in pediatric medical malpractice cases are (1) cardiac or respiratory arrest, (2) appendicitis, (3) disorders of male genital organs, (4) encephalopathy, and (5) meningitis.**

Pitfall | Failing to understand the three levels of a presenting complaint

The three levels of a chief complaint

Level 1 is the *patient's question*. For example, patients with chest pain will often have a symptom-centric concern: "Am I having a heart attack?" Addressing this patient question with data gathering (history and physical exam), testing (if indicated), medical decision making (MDM), and discharge instructions serves two purposes:
- It ensures the patient is confident that their main consideration has been evaluated and addressed.
- Reassurance is a part of healing. If this diagnosis is not occurring (i.e. chest pain in an 18-year-old with an obvious muscular strain), the patient will be able to cognitively offload their concern and be "reassured" that this diagnosis has been excluded.

Level 2 is the *presentation's question*. To continue our example of chest pain, whereas the *patient's* question is "Am I having a heart attack?" the *presentation's* question is "Am I having acute coronary syndrome, pulmonary embolism (PE), aortic dissection, or another serious diagnosis?" Data gathering should address these considerations and if they seem unlikely to be occurring, this can be discussed in the MDM note.

Level 3 is a consideration that we still have at the completion of the urgent care evaluation: Does *diagnostic uncertainty* remain? If this is the case, the patient should be made aware of this uncertainty, which would encourage them to be more likely to return if their symptoms progress, change, or do not resolve. Consider the case of a 39-year-old woman who presented with chest pain, had a diagnosis of acute bronchitis, was discharged, and then sustained a cardiac arrest that evening. The husband was so confident that a heart attack had been ruled out that when he called the emergency medical services (EMS) he told them that she "choked on a piece of chicken" [9].

Our "acceptable miss rate" is never zero, but if the serious diagnosis is very unlikely, it is essential to consider that patient harm could ensue from extraneous testing [3, 10].

Picking the low-hanging fruit

In this and the following sections we consider the most frequently missed diagnoses in adults and children, respectively, that are likely to result in legal implications.

The top five adult diagnoses

Cardiac or respiratory arrest

Pitfall | Failure to anticipate and plan for an arrest in the urgent care center

Though the management of an actual arrest in urgent care will be to call for EMS, there are two ways in which we can improve the care of these patients: 1) anticipation of arrest and 2) training for management of the patient during an arrest.
- *Anticipation of arrest*: High-risk features include patients with significant tachypnea, tachycardia, hypotension, cyanosis, anaphylactic reactions, airway impingement, significantly altered consciousness, intoxication with decreased respirations, or an electrocardiogram (ECG) showing ST-elevation myocardial infarction (STEMI), Mobitz II, or third-degree heart block. The goal will be to see these patients quickly and to call EMS immediately.
- *Care during arrest*: The most important part of excellent care during an arrest is to plan for the possibility and procedures to follow during an arrest. Having staff trained in basic life support, a team leader to manage

rotation of roles (such as two minutes of compression then changing), ensuring adequacy of compressions (timing and depth), use of an automatic external defibrillator (AED), providing ventilations, and, in select situations, administration of intranasal naloxone will increase the effectiveness of cardiopulmonary resuscitation (CPR).

Acute myocardial infarction

An acute myocardial infarction may present in multiple fashions, including chest discomfort and, in certain populations such as women, those with diabetes, and the elderly, with dyspnea, diaphoresis, or even fatigue [11]. Asking patients with epigastric pain questions about exertional discomfort or associated symptoms will help to differentiate between gastritis and inferior myocardial infarction. If there is significant concern an ECG should be performed quickly, which can expedite the diagnosis and transfer to an ED; however, a normal ECG does not rule out myocardial infarction [12].

What can we do at the urgent care center? In patients who have ECG findings of ischemia or STEMI with a consistent clinical picture, oral administration of aspirin 325 mg should be done, unless there is an allergy. If hypoxemic with an oxygen saturation less than 90%, the patient should be administered oxygen. Ensuring there is a team leader to give a report to the paramedics can facilitate the rapid transfer of information. Calling ahead to the ED can facilitate preparation of a catheterization suite for patients with STEMI.

KEY FACT | **Acute coronary syndrome may present with dyspnea, diaphoresis, back or epigastric pain, fatigue, or reproducible chest wall pain. Have a low threshold for obtaining an ECG.**

Aortic aneurysm

Pitfall | Failing to consider abdominal aortic aneurysm in patients over the age of 60 with back pain

Abdominal aortic aneurysm (AAA) is common. It occurs in 1% of patients 55–64 years of age and increases by 2–4% per decade. The risk of rupture for a 5 cm AAA is 5% per year, whereas an AAA over 6 cm has a yearly rupture rate of 10–50% [13].

A missed AAA represents a significant risk in urgent care medicine. Often patients will present with back pain, which may be confused with a simple lumbosacral strain. In fact, most ruptured AAA patients present atypically and not with the classic triad of a pulsatile abdominal mass, abdominal or back pain, and hypotension [14]. Suspicion for AAA should be higher in patients over the age of 60 who present with back pain, but without a mechanism, clear etiology, or pain with range of motion or palpation.

Evaluation with bedside ultrasound approaches 100% sensitivity to exclude an AAA [15]. Although this is not common in urgent care practice currently, it has the potential to improve patient care and safety.

A review of 346 cases of litigation for AAA revealed that claims were most often for failure to diagnose (61%), followed by delayed diagnosis and treatment (21%). It also showed that AAA often presented with abdominal pain (63%) and back pain (37%) [16].

These authors recommend consideration of AAA in age-appropriate (especially over the age of 60) patients with back pain without a clear mechanism of injury, without subjective or objective evidence of muscular pain, and without another clear etiology. Additionally, an MDM note should be written specifically detailing why the clinician does *not* think this diagnosis is present.

KEY FACT | **An abdominal aortic aneurysm may be falsely attributed to a lumbosacral strain. Consider this in the differential if there is not a mechanism of strain, subjective pain (with range of motion), or objective pain (with palpation).**

Pulmonary embolism

Pitfall | Failure to consider pulmonary embolism in patients who have recently had surgery or orthopedic trauma

PE is a potentially difficult diagnosis. Symptoms can mimic an upper respiratory infection, anxiety, or musculoskeletal chest wall pain.

Wilson *et al*. evaluated 277 cases of litigation in the study "Common reasons for malpractice lawsuits involving pulmonary embolism and deep vein thrombosis." This study found that the most common reason for litigation was "failure to diagnose and treat" (62%),

with the most common etiology being prior surgery (41%), followed by orthopedic trauma (31%) [17].

The pulmonary embolism rule-out criteria (PERC) rule can decrease unnecessary testing in applicable populations. If a patient meets all the criteria and the clinician's pretest probability is <15%, the risk of PE is only 1.4%, which is low enough to safely discharge without further workup [18, 19]. The PERC rule has eight elements:

- Age <50 years.
- Heart rate <100 bpm.
- Oxygen saturation on room air ≥95%.
- No unilateral leg swelling.
- No hemoptysis.
- No recent surgery (general anesthesia) or trauma within four weeks.
- No prior PE or deep vein thrombosis.
- No oral contraceptives, hormone replacement, or estrogen hormones (in males or females).

The most effective ways to prevent missing PE and a subsequent lawsuit are to:

- Consider PE in the differential.
- Use a clinical decision rule such as the PERC rule in applicable populations.
- Record an MDM note to explain that PE was considered and why it is unlikely to be the cause of the patient's symptoms.

KEY FACT | **A pulmonary embolism can be safely excluded with a negative PERC assessment if the clinician's pretest probability is assessed at <15%.**

Appendicitis

One of the primary concerns in patients with right lower quadrant (RLQ) pain is appendicitis. It is essential to specifically address this concern in the data gathering, the MDM, and the discharge instructions.

Symptoms will vary between patients and infrequently present as in the textbook [20]:

- Fever is not specific or sensitive, and a patient can have appendicitis without a fever.
- RLQ pain is present over 90% of the time, but that leaves 10% of patients presenting without RLQ pain. The classic location for appendicitis is McBurney's point, which is one-third of the way from the anterior super iliac spine to the umbilicus.
- Vomiting only occurs 75% of the time.
- Migrating abdominal pain only occurs 50% of the time.
- Rovsing's sign (pain in the RLQ with palpation in the left lower quadrant) is not sensitive, but it has a specificity of 58–96%.
- Check for and document rigidity, rebound, and guarding, which are more specific than sensitive.

The expanded differential for RLQ pain for both women and men should include zoster, incarcerated hernia, and ureterolithiasis. Additionally, differentials should be considered based on male and female genitourinary anatomy. In women, consider ectopic pregnancy, ovarian torsion, tubo-ovarian abscess, and ovarian cyst. In men, consider testicular pathology including torsion.

KEY FACT | **When the patient with RLQ pain is discharged from urgent care, ensure:**

- **They are aware of and understand the diagnostic uncertainty (appendicitis has not been 100% excluded).**
- **They know that they should return if they have any pain whatsoever in 12 hours.**
- **They know that they should return *immediately* with increased pain, fever, or vomiting.**

The top five pediatric diagnoses

Cardiac or Respiratory arrest

The pediatric patient with impending arrest is like a four-alarm fire in the urgent care center. Tips to localize patients who are at risk include tachypnea, tachycardia, hypoxemia, cyanosis, altered consciousness, and anaphylaxis or airway obstruction.

Pointers for localizing patients with impending arrest include:

- Undressing the patient to check for intercostal or subcostal retractions and signs of trauma or nonaccidental trauma.
- Flagging grossly abnormal vital signs.
- Assessing for airway compromise in patients with stridor, drooling, or those placing themselves in a "tripod" position (tripoding).

Most of the time the urgent care approach will be to localize patients at impending risk and alerting EMS. However, there is an ability to mitigate arrest in urgent care with the use of albuterol for an asthma exacerbation, administering intramuscular (IM) epinephrine for anaphylaxis, initiating intravenous (IV) fluids in patients with significant hypovolemia, or providing oxygen for patients with respiratory compromise or status epilepticus.

KEY FACT | **Pediatric patients have the ability to compensate for serious illness with tachycardia and tachypnea. If there are signs of hemodynamic instability, consider rapid transfer before the arrest occurs.**

Appendicitis

Pitfall | Failing to consider that the three main factors in missed appendicitis are female sex, multiple comorbidities, and constipation

A review of 234 cases of alleged malpractice found that the primary reason for legal action was failure to diagnose (67%) [21]. The diagnosis can never be made unless it is considered in the differential. The presentation of appendicitis is not always the classically described periumbilical pain migrating to McBurney's point with associated anorexia, vomiting, and fever [22].

A study with over 22,000 children localized three main factors associated with a missed diagnosis: 1) female sex, 2) multiple comorbidities, and 3) associated constipation [23]. It is important to avoid "anchoring" when a parent suggests constipation as the cause [24]. An x-ray showing constipation can be misleading, and despite extensive efforts practice has been difficult to change [25]. The authors recommend *against* obtaining plain film abdominal x-rays in the urgent care center unless a foreign body is suspected.

A large meta-analysis and systemic review revealed that factors associated with the highest likelihood ratio (LR) were migration of pain to the RLQ, pain when the patient was asked to cough or hop, and Rovsing's sign (LR 3.5) [26]. However, none of these findings is definitive enough to make or rule out the diagnosis.

KEY FACT | **An x-ray showing "constipation" does not exclude the diagnosis of appendicitis.**

Disorders of male genital organs

Testicular torsion can be tricky as the physical exam is not diagnostic. The differential includes nonsurgical causes of testicular pain such as epididymitis, epididymo-orchitis, hydrocele, or varicocele. Additionally, the testicle may torse then detorse, resulting in an improvement in pain. Finally, and even more troubling, up to 31% of the time testicular torsion may present with isolated abdominal pain [27].

Both Gaither *et al*. and Colaco *et al*. looked at over 100 cases of litigation involving testicular torsion. They found that the most successful suits are against emergency physicians (and not against urologists), that atypical presentation is common in malpractice cases, and that ultrasound is not always adequate to exclude torsion. Both recommend urologic consultation in cases of testicular pain [27, 28].

KEY FACT | **Consider a "cookbook" approach to evaluation of testicular pain where almost all patients obtain an emergency ultrasound and urologic consultation.**

Encephalopathy and meningitis

It may be easy to let our guard down for patients with a presumed viral syndrome who also have cephalgia or for the patient with chronic headaches who "felt warm" at home.

For the patient with typical features of meningitis such as fever, severe headache, altered consciousness, petechial rash, and vomiting, it would be unlikely to miss this diagnosis. However, sometimes the patient's symptoms are vague. For example, a study by McAbee *et al*. revealed that the top five presenting symptoms of meningitis were fever (74%), nausea/vomiting (49%), lethargy (32%), headache (27%), and influenza symptoms (25%) [29]. Only 12% had altered consciousness and only 10% had a stiff neck. In patients with meningitis that was initially missed, the diagnosis was erroneously documented as viral infection/influenza 36% of the time.

KEY FACT | **Beware of patients with lone headache and fever (without symptoms suggesting an alternative diagnosis such as generalized myalgias, rhinorrhea, vomiting, or other infectious source).**

Bringing it all home

Pitfall | Failure to involve the patient in the medical decision-making process

Let us return to the initial scenario of the 23-year-old woman with chest pain and hemoptysis, with *some* risk for PE but a viable alternative explanation for her symptoms. She cannot be excluded with the PERC rule as she is using oral contraceptives, leaving consideration of PE

to the bedside clinician and the patient. This is where shared decision making (SDM) comes into play.

SDM is a decision between multiple viable and safe pathways undertaken by the clinician and the patient in partnership. However, there is one large caveat: SDM must be performed with the *guidance of the clinician* and not left only to the patient. SDM can occur when there is adequate time for the decision to be made by a patient who is competent to make the choice [30, 31].

An experimental study of 737 emergency physicians found that about two-thirds of participants agreed that using an SDM model would decrease their medical legal risk. This was based on the idea that SDM with the patient gives physicians *permission* to practice good medicine, with the insinuation that this could result in the best care for the patient, and potentially decrease the practice of defensive and possibly harmful medicine [30, 32].

How would we document SDM in the MDM note with the initial scenario presented at the beginning of the chapter? One potential approach is as follows: "Though there is an increased risk of pulmonary embolism with the patient's use of oral contraceptives, this diagnosis seems unlikely, based on the fact that there is no pain or swelling of the lower extremities, no tachypnea, tachycardia, or hypoxemia. Additionally, there is a viable explanation for the patient's symptoms, as the hemoptysis started after a vigorous episode of coughing. The patient understands that there is a very small amount of diagnostic uncertainty remaining and will return if her symptoms progress, worsen, or do not resolve. She expresses understanding of diagnostic uncertainty and the return precautions. She agrees to the discharge plan and will follow up with primary care in the next two or three days."

Conclusion

The balance between good medical care and considerations of our own legal risk is a real and constant presence. Using approaches presented in this chapter for the "low-hanging fruit" in relation to common medical malpractice symptoms and diagnoses should result in not only the best care for our patients but also decreased medical malpractice risk for clinicians.

Pearls for improving patient outcomes

- An inferior myocardial infarction may present as epigastric pain.
- Include pulmonary embolism in the differential in patients with recent orthopedic procedures or trauma.
- One-quarter of patients with meningitis present with influenza symptoms.
- Inform the patient if there is diagnostic uncertainty at the completion of the evaluation.
- Reassurance (that a more serious disease is not occurring) is a part of healing.

References

1 Sakhnini A, Bisharat N. Practice behavior of emergency department physicians caring for patients with chest pain. *Am J Emerg Med* 2019; **37**(6): 1210–1212. https://doi.org/10.1016/j.ajem.2018.11.039.

2 Pines JM, Isserman JA, Szyld D, *et al*. The effect of physician risk tolerance and the presence of an observation unit on decision making for ED patients with chest pain. *Am J Emerg Med* 2010; **28**(7): 771–779. https://doi.org/10.1016/j.ajem.2009.03.019.

3 Weinstock MB, Mattu A, Hess EP. How do we balance the long-term health of a patient with the short-term risk to the physician? *J Emerg Med* 2017; **53**(4): 583–585. https://doi.org/10.1016/j.jemermed.2017.06.010.

4 Ong S, Nakase J, Moran GJ, *et al*. Antibiotic use for emergency department patients with upper respiratory infections: prescribing practices, patient expectations, and patient satisfaction. *Ann Emerg Med* 2007; **50**(3): 213–220. https://doi.org/10.1016/j.annemergmed.2007.03.026.

5 Fenton JJ, Jerant AF, Bertakis KD, *et al*. The cost of satisfaction: a national study of patient satisfaction, health care utilization, expenditures, and mortality. *Arch Intern Med* 2012; **172**(5): 405–411. https://doi.org/10.1001/archinternmed.2011.1662.

6 Rothberg MB, Sivalingam SK, Ashraf J, *et al*. Patients' and cardiologists' perceptions of the benefits of percutaneous coronary intervention for stable coronary disease. *Ann Intern Med* 2010; **153**(5): 307–313. https://doi.org/10.7326/0003-4819-153-5-201009070-00005.

7 Wong KE, Parikh PD, Miller KC, *et al*. Emergency department and urgent care medical malpractice claims 2001–2015. *West J Emerg Med* 2021; **22**(2): 333–338. https://doi.org/10.5811/westjem.2020.9.48845.

8 Glerum KM, Selbst SM, Parikh PD, *et al*. Pediatric malpractice claims in the emergency department and urgent care settings from 2001 to 2015. *Pediatr Emerg Care* 2021; **37**(7): e376–e379. https://doi.org/10.1097/PEC.0000000000001602.

9 Alblaihed L, Cobert E, Mattu A. A 39-year-old-woman with multiple complaints: coughing during cold & flu season. In: Weinstock MB (ed.), *Bouncebacks! Medical and Legal*. Columbus, OH: Anadem Publishing; 2024: case 1.

10 American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Suspected Non–ST-Elevation Acute Coronary Syndromes, Tomaszewski CA, Nestler D, *et al*. Clinical policy: critical issues in the evaluation and management of emergency department patients with suspected non-ST-elevation acute coronary syndromes. *Ann Emerg Med* 2018; **72**(5): e65–e106. https://doi.org/10.1016/j.annemergmed.2018.07.045.

11 Canto JG, Goldberg RJ, Hand MM, *et al*. Symptom presentation of women with acute coronary syndromes: myth vs reality. *Arch Intern Med* 2007; **167**(22): 2405–2413. https://doi.org/10.1001/archinte.167.22.2405.

12 Welch RD, Zalenski RJ, Frederick PD, *et al*. Prognostic value of a normal or nonspecific initial electrocardiogram in acute myocardial infarction. *JAMA* 2001; **286**(16): 1977–1984. https://doi.org/10.1001/jama.286.16.1977.

13 Aggarwal S, Qamar A, Sharma V, *et al*. Abdominal aortic aneurysm: A comprehensive review. *Exp Clin Cardiol* 2011; **16**: 11–15.

14 Gaughan M, McIntosh D, Brown A, *et al*. Emergency abdominal aortic aneurysm presenting without haemodynamic shock is associated with misdiagnosis and delay in appropriate clinical management. *Emerg Med J* 2009; **26**: 334.

15 Concannon E, McHugh S, Healy DA, *et al*. Diagnostic accuracy of non-radiologist performed ultrasound for abdominal aortic aneurysm: systematic review and meta-analysis. *Int J Clin Pract* 2014; **68**(9): 1122–1129. https://doi.org/10.1111/ijcp.12453.

16 Choinski K, Sanon O, Tadros R, *et al*. Review of malpractice lawsuits in the diagnosis and management of aortic aneurysms and aortic dissections. *Vasc Endovasc Surg* 2022; **56**(1): 33–39. https://doi.org/10.1177/15385744211026455.

17 Wilson E, Phair J, Carnevale M, *et al*. Common reasons for malpractice lawsuits involving pulmonary embolism and deep vein thrombosis. *J Surg Res* 2020; **245**: 212–216. https://doi.org/10.1016/j.jss.2019.07.079.

18 Kline JA, Mitchell AM, Kabrhel C, *et al*. Clinical criteria to prevent unnecessary diagnostic testing in emergency department patients with suspected pulmonary embolism. *J Thromb Haemost* 2004; **2**(8): 1247–1255. https://doi.org/10.1111/j.1538-7836.2004.00790.x.

19 Kline JA, Courtney DM, Kabrhel C, *et al*. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. *J Thromb Haemost* 2008; **6**(5): 772–780. https://doi.org/10.1111/j.1538-7836.2008.02944.x.

20 Moris D, Paulson EK, Pappas TN. Diagnosis and management of acute appendicitis in adults: a review. *JAMA* 2021; **326**(22): 2299–2311. https://doi.org/10.1001/jama.2021.20502.

21 Choudhry AJ, Anandalwar SP, Choudhry AJ, *et al*. Uncovering malpractice in appendectomies: a review of 234 cases. *J Gastrointest Surg* 2013; **17**(10): 1796–1803.

22 Weinstock MB, Jolliff H. High-risk medicolegal conditions in pediatric emergency medicine. *Emerg Med Clin North Am* 2021; **39**(3): 479–491. https://doi.org/10.1016/j.emc.2021.04.001.

23 Mahajan P, Basu T, Pai CW, *et al*. Factors associated with potentially missed diagnosis of appendicitis in the emergency department. *JAMA Netw Open* 2020; **3**(3): e200612.

24 Croskerry P. Achieving quality in clinical decision making: cognitive strategies and detection of bias. *Acad Emerg Med* 2002; **9**(11): 1184–1204. https://doi.org/10.1111/j.1553-2712.2002.tb01574.x.

25 McSweeney ME, Chan Yuen J, Meleedy-Rey P, *et al*. A quality improvement initiative to reduce abdominal X-ray use in pediatric patients presenting with constipation. *J Pediatr* 2022; **251**: 127–133. https://doi.org/10.1016/j.jpeds.2022.07.016).

26 Benabbas R, Hanna M, Shah J, *et al*. Diagnostic accuracy of history, physical examination, laboratory tests, and point-of-care ultrasound for pediatric acute appendicitis in the emergency department: a systematic review and meta-analysis. *Acad Emerg Med* 2017; **24**(5): 523–551.

27 Gaither TW, Copp HL. State appellant cases for testicular torsion: case review from 1985 to 2015. *J Pediatr Urol* 2016; **12**(5): 291.e1–291.e5.

28 Colaco M, Heavner M, Sunaryo P, *et al*. Malpractice litigation and testicular torsion: a legal database review. *J Emerg Med* 2015; **49**(6): 849–854.

29 McAbee GN, Donn SM, Mendelson RA, *et al*. Medical diagnoses commonly associated with pediatric malpractice lawsuits in the United States. *Pediatrics* 2008; **122**(6): e1282–e1286.

30 Probst MA, Kanzaria HK, Frosch DL, *et al*. Perceived appropriateness of shared decision-making in the emergency department: a survey study. *Acad Emerg Med* 2016; **23**(4): 375–381. https://doi.org/10.1111/acem.12904.

31 Probst MA, Kanzaria HK, Schoenfeld EM, *et al*. Shared decision making in the emergency department: a guiding framework for clinicians. *Ann Emerg Med* 2017; **70**(5): 688–695. https://doi.org/10.1016/j.annemergmed.2017.03.063.

32 Schoenfeld EM, Mader S, Houghton C, *et al*. The effect of shared decisionmaking on patients' likelihood of filing a complaint or lawsuit: a simulation study. *Ann Emerg Med* 2019; **74**(1): 126–136. https://doi.org/10.1016/j.annemergmed.2018.11.017.

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